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Proceedings

of the

Fifth Annual American Association

of

Equine Practitioners Convention

Chicago, Illinois
DECEMBER, 1959

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of the

Fifth Annual American Association

of

Equine Practitioners Convention

Held at

LA SALLE HOTEL

Chicago, Ill.

December 14 thru 16

1959

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FOREWORD

This is the fourth annual proceedings book of the American Association of Equine Practitioners covering the fifth annual convention held in Chicago, Illinois, December 14 through 16, 1959.

The entire proceedings of this convention were transcribed and are a matter of record. This booklet is confined to the Scientific papers and Panels plus the Open forum for purposes of brevity and economy.

Doctor S. F. Scheidy, President of the American Veterinary Medical Association delivered the major address at the annual banquet before the entire membership in attendance during the convention.

JORDAN G. WOODCOCK, D.V.M.
President-Elect and
Program Chairman

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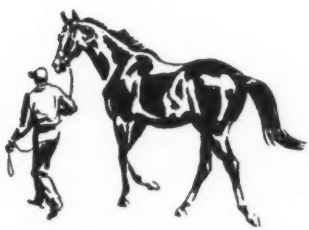

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Banquet Address to American Association of Equine Practitioners

"WHO ARE WE?"

S. F. Scheidy, V.M.D., President of A.V.M.A.

The veterinary profession in the United States and Canada has expanded so rapidly in recent years that it would seem worthwhile at this time to pause and take stock, to try to answer the question of just who we are in terms of numbers, background of training, types of services we offer, facilities at our command, and the kind of organized effort we have sponsored in order to improve our services and benefit the profession generally. This paper is an effort to answer that question by means of both statistics and generalized summary. It considers in turn the present student veterinary population, graduate membership in the profession, and the membership, scope, and plans of the American Veterinary Medical Association, which is, of course, the central agency of communication for those in veterinary practice.

In September of 1959, a veterinary school was established on the campus of Purdue University, Indiana, with a beginning enrollment of 50 students. This brought to twenty the number of veterinary schools and colleges in the United States and Canada. Recently we sent a brief questionnaire to the deans of these schools. Our purpose was to obtain certain general information about the student body in each institution within a given period of time, the school year of 1957-58 being selected. Table I summarizes the data which we received from eighteen schools. Of the total of 3,695 students in school that year, 97.5% were boys and 2.5% were girls. The average age was 23.5, and 43.8% were married, a figure that certainly has changed since our student days. It is interesting to observe that the split was almost even between those who came from farms and those who came from urban areas. Fully 60% had received more than the minimum two years of pre-professional college training, 30% with three years and another 30% with four years or more. This added preparation certainly stands the student in good stead when he comes up against the rigid curriculum requirements found in veterinary colleges nowadays.

What happens to students immediately upon graduation may be a good index of their primary interests. Table II summarizes the data we received from the graduating classes of 1958. While we did not obtain information about post-graduate activities from all of those who completed their veterinary training in 1958, the tabulation does seem to reflect accurately the general trend of recent years. Compared to the situation of ten or fifteen years ago, fewer graduates today (21% of the 1958 group) are entering their own private practice within the first year after leaving

veterinary school; a much larger number (62%) are joining others either as partners or as assistants. Several factors are probably at work here: one, they may not be financially able to start their own practice; two, and more important, many of them feel the need for experience with an established practitioner before launching out on their own. They, thus, enter upon what I would choose to call a self-imposed internship. Then, of course, a certain number (6.5%) found opportunities in government positions, either at the federal, state, municipal, or county levels. A relatively large number (9.5%) entered military service, receiving officers' commissions almost without exception. The number of those going on to postgraduate studies (6% of the 1958 group) seems to be steadily increasing; we are happy about this since it opens further opportunities in the fields of research and teaching. In this connection, I should like to point out that there are increasing requests from graduate students for financial assistance from agencies like our A.V.M.A. Research Fund. Unfortunately, we do not have sufficient funds to provide loans for all deserving candidates; it is highly desirable, therefore, that we make a greater effort in soliciting additional gifts and bequests. Surely we will need more and better trained individuals in our teaching and research institutions.

Following the collection of data for the 1958 A.V.M.A. Directory, we prepared a summary of the different types of professional service in which the graduate veterinarians were engaged. As Table III indicates, just about 50% of the 20,861 veterinarians in the United States and Canada are engaged in general practice, while 15% are in special practice. The number in teaching and research (5%) will surely grow with the increase in schools and in research demands. The number in commercial enterprises — pharmaceutical, biological, dairy meat packing industries — is relatively very low, as is the number of those who have taken positions outside the field of veterinary services. About 75% of the veterinarians in the United States and Canada belong to the American Veterinary Medical Association, which is actually a federation of sixty-three associations, representing fifty states, Canadian provinces, United States territories, federal government and the Armed Forces. Each constituent association elects a delegate to the A.V.M.A. House of Delegates. In addition, there are thirty-nine specialty organizations, including the American Animal Hospital Association, which is one of the first and largest of the member groups, the American Association of Veterinary Nutritionists, American Association of Women Veterinarians, American College of Veterinary Pathologists, American Association of Equine Practitioners, American Board of Laboratory Animal Medicine, and, still in a formative stage, the American Board of Veterinary Radiologists. Anatomists, physiologists, pharmacologists, parasitologists, and others are beginning to form organizations, an encouraging development, paralleling the ad-

vances which our sister organization, the American Medical Association, has made over the years.

The House of Delegates is the legislative body of the Association and meets annually. The Executive Board is the administrative branch and consists of elected representatives from the twelve districts in the United States and Canada as well as the past presidents, president, and president-elect. This body usually convenes twice each year in order to deal with the affairs of the Association. In the intervening periods, the Board of Governors, consisting of the president, president-elect, and the chairman of the Executive Board, in cooperation with the central office in Chicago, manages the operation of the Association.

Much of the planning, service, and research work with respect to the problems of the Association is done by members of the various councils. Currently, there are six councils (Judicial, Education, Research, Veterinary Services, Public Health and Regulatory Veterinary Medicine, and Biological and Therapeutic Agents). The members are elected by the Delegates to the House for five-year terms.

What are the membership benefits in the A.V.M.A.? In the first place, the Association publishes a semi-monthly journal, a bi-monthly research journal, and a bi-annual directory, as well as other literature of use and of interest to students and members. There is also a film and slide library maintained in the central office for the convenience of members. Such materials can be obtained at a nominal cost by them for professional use. An active insurance program is available for members, including liability insurance as well as group health, accident and life insurance at very reasonable cost to subscribers. This service, rather new in the Association, presently enrolls members from about thirty states.

Annual meetings or conventions are a major activity of the Association. These are held in five different sections or zones of the United States and Canada. The general convention, frequently held in August, usually lasts five days. In 1960, it will be held in Denver. There are many other, more indirect benefits. Public relations and publicity activities are now administered by full-time employees from the central office in Chicago. Participation in such programs as the National Science Fair, distribution of releases to constituent associations, assistance in preparing articles for publication in farm and general magazines, preparation of booklets and pamphlets for distribution to the public — all these services are available to members.

An expanded professional relations program furnishes many day to day services to members. Last autumn, a two-day conference of Constituent Association Secretaries was held in Chicago and was attended by more than forty official representatives and delegates, officers of the A.V.M.A., and staff personnel. The A.V.M.A. Washington office is now a well-functioning organiza-

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tion; much valuable information is obtained there which is published regularly in the J.A.V.M.A. Good and active relations with federal agencies are very important and should have been initiated years ago.

The A.V.M.A. Research Fellowship program, begun some twenty years ago, has grown considerably over the years. At the same time, interest in postgraduate education has greatly increased, and we now have many more requests for financial aid to graduate students than we can satisfy. Recently, the Executive Board took definite steps to more actively solicit funds for the Research Trust, and we hope soon to employ a person who will devote all or most of his time to this worthy project. Arrangements have also been made whereby we can receive gifts as memorial funds.

The A.V.M.A. is a federation of associations so organized that veterinarians throughout the United States and Canada — people with a variety of professional interests — are given an opportunity as well as the responsibility to administer the affairs of this international body of professional men and women.

The interests of students, graduates, and the general public with respect to the animal population and public health are given consideration. It is in this area that such a body can and must serve a useful purpose. The Association will soon be celebrating its centennial (1963), and history reveals that it has indeed served the people well in these two neighboring countries. Rapid growth in the number of veterinarians especially in the past fifteen to twenty years has been paralleled by a marked increase in the membership of the A.V.M.A. As of December 31, 1959, there were 14,862 members in the Association. With this growth, increased services have been provided. Upgrading of schools and colleges, increased postgraduate programs for veterinarians, as well as the greatly enlarged professional service rendered by veterinarians to the public are in keeping with the times. The indications are that a bright future is ahead for the profession, and all we need to do is to further improve ourselves and the service that is to be expected from us.

Table I

Students enrolled for the period	3695	
Male students	97.5%	
Female students	2.5%	
Average age	23.5	years
Married	43.8%	
From farms	47.7%	(out of 1930)
From urban areas	52.2%	(out of 1930)
Two years	40%	
Three years	30%	
Four years or more	30%	

Table II

Entered own private practice	21.6%	(out of 633)
Employed in practice	61.6%	(out of 633)
Government service (civilian)	6.5%	(out of 633)
Military service	9.5%	(out of 633)
Postgraduate studies	6.0%	(out of 729)

Table III

General practice	50%	
Special practice	15%	
Teaching and/or Research	5%	
Government service	17.5%	
Commercial	1.5%	
Inactive or retired	5.0%	
Other classification	1.0%	
Unknown classification	5.0%	
	100.0%	(20.861)

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FEDERAL REQUIREMENTS FOR INTERNATIONAL AND INTERSTATE SHIPMENTS OF EQUINES

B. C. Swindle, D.V.M.

The inspection and certification of imported equines is one of the responsibilities of the Animal Inspection and Quarantine Division, Agricultural Research Service, U. S. Department of Agriculture. With the ever-increasing number of air shipments of livestock, the port veterinarian, when making health inspections, must be more alert than ever before. In only a matter of hours animals may arrive in this country from any country in the world, many of which are infected with diseases that are not present in the United States.

All equines, ruminants and swine are examined for health aboard the carriers at the first port of entry by a Departmental veterinarian. In addition, we are also on the lookout for prohibited animal byproducts, hay and straw that may be contaminated with viruses or with ticks which would cause staggering losses to our livestock industry if introduced into this country.

No prior permit is required for the importation of horses. However, horses offered for entry from any part of the world except Canada, Central America and the West Indies must be accompanied by a certificate of a salaried veterinary officer of the national government of the country of origin showing that the animals described in the certificate have been in the said country for the preceding 60 days and that as far as it has been possible to ascertain no case of dourine, glanders, surra, epizootic or ulcerative lymphangitis has occurred in the locality where the horses have been kept during such period.

Horses imported from any country in the world except Great Britain, Ireland, Australia, New Zealand and Canada must be subjected to a blood test upon arrival at the port of entry. This blood sample is taken by a practicing veterinarian under the supervision of a veterinarian in the employ of the Animal Inspection and Quarantine Division and a serum sample is forwarded to the Animal Disease Station at Beltsville, Maryland, for a complement-fixation test for dourine and glanders. In the meantime, these animals making entry at the Port of New York are retained at the U. S. Animal Quarantine Station, Clifton, New Jersey, until the results of the test are known. Negative animals are released outright, and a Federal health certificate is issued. In the event a reactor is found, the importer has a choice of either destroying the animal or returning it to the country of origin.

Upon inspecting the animals at the port of entry and before

permitting them to land, the inspector may require their disinfection or the disinfection of accompanying equipment as a precautionary measure against foot-and-mouth disease or any other disease dangerous to the livestock of the United States. The hay, straw, or other forage accompanying the shipment may remain aboard the carrier or be incinerated under supervision.

Horses imported from Mexico, Central America, West Indies, and other tick-infested countries are closely inspected for ticks upon arrival. If ticks are found, entry is prohibited and the shipment returned to the country of origin. If no ticks are found upon inspection, the horses are dipped or sprayed with an arsenical solution and if further health requirements are met they are released outright.

In addition to examining the horses and accompanying documents regarding health status, we are also charged with the responsibility of examining animals for certification when entered duty-free for breeding purposes. This examination is rather time-consuming and great care must be taken to record and check the exact age, sex, breed, color markings and other details pertinent to the importation.

At the port of New York during the past twelve months ending November 30th, we handled 371 importations consisting of 1,079 horses, of which 253 importations consisting of 394 animals were imported by air, while 118 shipments consisting of 685 horses were imported by sea. Probably, half of the horses imported were entered duty-free for breeding purposes.

All horses intended for exportation to a foreign country should be accompanied to the port of export by a certificate of health issued by an accredited veterinarian and endorsed by a veterinarian in charge of the Animal Disease Eradication Division of the state of origin. These certificates should show proper identification of the animals in the shipment with respect to age, sex, breed, color, and when tattoo markings are present, they should be included. If no tattoo markings are available then the outstanding color markings should be indicated. Each animal must be identified to the extent that the veterinarian at the port of export is positive that the horse presented is the same as the one covered by the accompanying health certificate.

Many countries require mallein and other tests. However, we find it impossible to keep abreast of the requirements of all countries. Exporters should obtain the required information by making inquiry at the Consulate representing the country of destination. Any additional tests or certifications should be indicated on the general health certificate when issued by the accredited veterinarian.

When the animals are presented at the port of export and found to be in a healthy condition and the accompanying certificates are in order, an Export Certificate is issued by the Animal

Inspection and Quarantine Veterinarian. Those animals that do not fully meet the requirements or cannot be positively identified are rejected for export by the Division Veterinarian. It is the policy of the Department of Agriculture to insure as far as possible that all animals exported from the United States have not been exposed to and are free of any infectious or contagious disease.

Regulations administered by the Animal Inspection and Quarantine Division also require that the transporting carriers provide proper space, fittings, ventilation, water and feed for the animals enroute, in order that they may be maintained and handled in a humane manner. The regulations are very exact as to the amount of space per animal, the type and construction of the stalls, and the humane care and handling of animals while in transit.

At the port of New York during the past twelve months ending November 30th, there were 96 shipments consisting of 248 horses exported to foreign countries, of which 62 shipments consisting of 108 horses were shipped by air, and 34 shipments consisting of 140 horses were shipped by sea.

Generally speaking, horses are acceptable in all states when shipped interstate, when accompanied by an official health certificate stating that the animals are free of infectious or contagious diseases or exposure thereto. However, it is not unusual, when an outbreak of some infectious or contagious disease occurs, for the adjoining states to require special health certification covering animals leaving the infected areas or state.

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PANEL DISCUSSION OF RACING'S 48-HOUR STIMULATION RULE

Moderator: JORDAN WOODCOCK, V.M.D.

Panel: MARSHALL CASSIDY

JAMES H. INGLIS
THADDEUS B. BRUNO
Y. T. OESTER, M.D.
JOHN MANFUSO
WILLIAM O. REED, D.V.M.
M. B. TEIGLAND, D.V.M.

MODERATOR WOODCOCK: Gentlemen, the panel discussion that will take place this afternoon is not the product of something that occurred only yesterday, so to speak, but one that, in my association with racing, has been a problem for many years. It has at least come to the forefront and was made so last year by Commissioner Inglis when he addressed this organization, and said he thought it would be helpful if this organization could help in the formulation of a rule which erroneously, in my opinion, is called the stimulation rule.

All during the past year various members of this organization have kept this in mind, have delved into the situation, have asked questions. I, for one, have questioned many of my associates, and men who are stewards in various sections of the country. We have come to the place where we must sit down and discuss the existing conditions with reference not only to the racing authorities who attempt to make racing as clean as possible, but go all the way down the line and include everybody concerned with racing.

The purpose of the panel this afternoon is to bring to light some of the problems that are involved. Perhaps from the discussion which you will hear this afternoon, constructive steps toward the end in mind, which would be a universal rule, will finally take place.

My procedure this afternoon will be to ask each member of the panel for a short discussion on his views and his thoughts along these lines. After the entire panel has had an opportunity to bring forth their particular thoughts, we will open the discussion to the floor.

I would then like to start with Mr. Cassidy. Let us have your views, sir.

MR. MARSHALL CASSIDY: I can only tell you that my

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experience in racing has been quite a number of years. I have been in racing fifty years. I was a steward, when the first rule was promulgated to deal with the so-called stimulation problem.

At that time, Mr. Widener, who was the President of the Miami Jockey Club at Hialeah sent Dr. Catlett and Charley Morgan, the chemist, to France to study the methods of analyzing saliva to determine whether a horse had been administered a stimulant or drug.

At that time, the country was rather inexperienced in promulgating such a rule, or enforcing it after it was established.

I was against the broad field that they started with, that is, with any administration of a drug, a trainer would arbitrarily be suspended for a minimum of sixty days. I thought that we should start with narcotics which had a deterimental effect on the animal and, if we were able to grow to the point where we could determine the presence of other drugs, extend it to wherever it might prove practical.

However, the rule was first embracive of everything that could be given to a horse, that was not food and water. Since that time there has been a lot of discussion at the various conventions of the National Association of Racing Commissioners. The rules have been changed by individual commissions to suit conditions they thought were proper. They finally have arrived with three or four variations.

A rule that is gaining favor, and much to my distaste, is one that prohibits the administration of a drug forty-eight hours before a race. I do not believe it is possible to determine whether an administration was made within forty-eight hours, and not fifty hours.

As a steward, or one of the group that is in control of that situation, from a racing standpoint, I felt that we were working with a rule that was impossible to properly administer.

We have in New York a rule that does not specify a time limit.

The rule reads: if the stewards shall find that any drug, stimulant or narcotic has been administered, or attempted to be administered internally or externally to a horse before a race, which is of such a character as could affect the racing condition of the horse in such race, such stewards shall impose such penalty and take such other action as they may deem proper under any of the rules of racing, including reference to the stewards of the Jockey Club, every person found by them to have administered, or to have attempted to administer or to have caused to be administered, or to have caused an attempt to administer, or to have conspired with another person to administer such drug, stimulant or narcotic.

It goes on to say the trainer, groom and any other person having charge, custody or care of the horse, are obligated to prop-

erly protect the horse and guard it against such administration or attempted administration, and if the stewards shall find that any such person has failed to show proper protection and guarding of the horse, they shall impose such penalty and take such other action as they may deem proper under any of the rules of racing, including reference to the stewards of the Jockey Club.

Although this rule speaks of "any drug, stimulant or narcotic" there still is quite a question in my mind as to the difference between a drug and medication such as hormones or vitamins which are given to a horse to improve his health.

I do not believe we will ever have a satisfactory rule unless veterinarians and horsemen are able to give to horses such aid as they deem necessary to provide a healthy animal to race.

I know it would be pretty difficult to retreat from the broad field that we have in prohibiting medicine, because of the reaction of the public. If we were to say at this time that we were going to change the rule, and only punish a person for the administration of narcotics, I think racing would lose a great deal by it.

However, in my opinion, narcotics are the most important item of which we have to be careful. I think that the stewards, if they are experienced, are able to control malpractices because of the evidence of inconsistent racing, particularly where the inconsistency is indicated by the fluctuation in odds before a race is run.

In California, just recently, they adopted a rule. This is dated December 9, 1959. It is Rule 1865: "Administration of Drugs. The administration of any drugs, chemical or medicine, hypodermically, orally or in any other manner, to a horse within 48 hours of a race in which he is entered to run is prohibited. Any owner, trainer, veterinarian or other person violating the provision of this section may be fined, suspended or have his license revoked."

I think that could be pretty tough on any owner, trainer or veterinarian or anybody else if he had administered something to the horse for the sole benefit of his health. Still, this makes it mandatory that he be fined, suspended or have his license revoked.

They have another rule, 1865.2, "Administration of Phenothiazine. No person shall enter or permit a horse to run in a race, any horse to which has been administered the drug, phenothiazine, within two weeks of such administration."

I cannot understand how they can tell if it was two weeks or three weeks, or what it was.

Our New York Commission, on July 16, 1949, added the following definition to their rules. This is under Rule 10 (a). It says: "Drugs shall be deemed to include substances intended for use in the diagnosis, treatment, mitigation, cure or prevention of disease in man or other animals, and substances other than foods

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intended to affect the structure or any function of man or other animal."

That is very broad. I do not see how a person could ice a horse's leg and not be in violation of that rule. Basically, I am strongly opposed to any time limit on the administration of medication. I am in favor of leaving the task to the stewards to supervise it, to the extent that veterinarians could treat a horse properly without attempting to affect his racing speed or condition, and I am not sure that I know just how to do it. In fact, I am sure I don't know, but I think from this meeting some good may come.

MODERATOR WOODCOCK: I would like very much to have Commissioner Inglis give us his views, please. [Applause]

MR. JAMES H. INGLIS: Mr. Chairman, Members of the American Association of Equine Practitioners: I appreciate very much the opportunity to be here this afternoon and to represent the N.A.S.R.C. I feel that the remarks I made at this convention a year ago have certainly borne fruit, because in your program this year you have given very generously of your time to consider this very important problem of the 48-hour rule.

I am here primarily today to listen and not to talk because I want to learn the answer as to this thing. I do not want to give any preconceived ideas.

In general, I can say that the Racing Commissioners are in favor, at the present time, of a 48-hour rule. However, we are open-minded, and we want to learn. We want to find out particularly what the views of this organization are.

I would like to point out that increasingly states have been adopting this rule over the last five or six years. Two years ago at our convention in Miami, there was a proposal on the agenda to eliminate the 48-hour rule. This item on the agenda was voted down, which was an indirect way of saying that the convention voted in favor of a 48-hour rule.

Last spring at our convention in New York, the Uniform Rules Committee received a recommendation for a specific version of the 48-hour rule which could be adopted and recommended to all commissions as a model rule on this subject.

After considerable discussion and debate, the rule that was adopted reads as follows:

"No horse to which has been internally administered any drug, chemical, medicine or hypodermic injection whatsoever within 48 hours of the time of a race shall be eligible to start in any race, except when such administration is permitted upon the specific written authorization of the stewards and in the presence of a veterinarian designated by the commission."

Two loopholes in this rule should be specifically pointed out. One is the use of the word "internally" administered. This takes us completely out of the business of prohibiting a leg paint or

some iodine or mercurochrome, or whatever you might want to put on a cut or a sore. The second is, of course, the permission, the specific written authorization to administer medicine within the 48-hour time.

This exception was put into the rule particularly to take care of the problem of the bleeders, which it is unsafe to start unless they are given a coagulant.

Either this rule or another version of the 48-hour rule is now in effect in most of the states; New York and Kentucky being the outstanding examples where it was not adopted.

I would like to comment for a minute on Marshall Cassidy's remarks, and I think he gave a very temperate explanation of his position on it. I gather that his objection to this rule is somewhat similar to the objection that I have heard expressed by the chemists, which is that you cannot set a definite time limit and say that you can give anything before that but you can't give it after that.

I would like to point out that this recommended 48-hour rule has been recommended not as an amendment or a substitute to any portion of the corrupt practices rule. In fact, the corrupt practices rule which Mr. Cassidy read still stands on our rule book almost verbatim the way he read it, and we have added this medication rule as a part of our rule book which deals with entries. It is not a corrupt practices rule; it is a rule regarding the entry of horses.

With the corrupt practices rule intact, without amendment, and even with the addition of this 48-hour rule on medication, the trainer is held fully responsible for any positive test which comes back from the laboratory, regardless of when the forbidden drug was administered.

You say, "Well, why have a rule, then?" The reason for a rule is because there are types of medication which could not be construed as stimulating or affecting the speed of a horse in a race.

Personally, I feel that the recent trouble we have had with the new drug, butazolidin, is a perfect example of why the 48-hour rule is valuable. Here we have a new product; it is not particularly new to horsemen, perhaps; may be it is three years old or four years old, but, as far as Racing Commissioners are concerned, it was new this last summer. With this drug, we were confronted with all sorts of conflicting claims. We had horsemen coming to us saying, "This is nothing but aspirin" and other people telling us that it was a very potent product, that definitely was a pain-relieving drug. In fact, these butazolidin administrations are sometimes referred to as "pain-balls."

I say that, when you have new products coming on the market, such as butazolidin, where you have conflicting claims as to what the effect of it is, and whether it is prohibited or isn't

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prohibited, the 48-hour rule is a valuable thing to fall back on because you can simply say to the trainer, "Well, you had no business giving anything at all to that horse within 48 hours," and that settles the argument.

I know that many horsemen feel that the use of that particular drug is essential. In fact, the National Trainers Guild has formally petitioned our organization to legalize this drug. In one state, Colorado, it has been legalized. In Colorado, during this past summer, the use of this drug was permitted, but every trainer which used it had to go to the Racing Commission and say, "I want horses A, B and C placed on the butazolidin list." Those horses that were put on that list had to be administered butazolidin every time they raced. Tests were taken of those horses, regardless of whether they were winners or whether they finished last, and the trainer was liable to disciplinary action if that test failed to come up positive for butazolidin.

That gives you a little idea of the administrative headaches that might come into play if this drug were authorized all over the country. If you multiply that by two or three other new drugs that horsemen wanted to administer, and which we were not quite sure what the effect of them was, you can see that, instead of testing a winner from each race, we would end up testing most of the horses in the race.

So, let me conclude by saying on this subject of the 48-hour rule, our association is on record in favor of it. As far as this meeting is concerned, I am open-minded, in behalf of the N.A.S.R.C., and I am glad we have with us today Thad Bruno who is Chairman of the Corrupt Practices Committee of the N.A.S.R.C. Thank you. [Applause]

MODERATOR WOODCOCK: Thank you very much, Commissioner Inglis.

I would now like to call on Commissioner Thaddeus Bruno, who is Chairman of the Illegal Practices Committee of the National Association of Racing Commissioners. Mr. Bruno!

MR. THADDEUS B. BRUNO: Mr. President and Members of the American Association of Equine Practitioners: I am delighted to be here and have this opportunity not only to give you my views on the 48-hour rule but also to listen to those whom I know are publicly and professionally opposed to its use.

I am now in my third year as a member of the Illegal Practices Committee of the National Association, and I can tell you that every time we meet the 48-hour rule is a subject of heated discussion. It is one of the most controversial rules that I have encountered in racing, and I am not prepared to stand here and tell you that it is a perfect rule. But, I think, to approach the problem, we should almost approach this problem of shall there or shall there not be a 48-hour rule, clinically.

Before we can discuss whether it is a good rule or a bad rule,

we should try to set up the problems which it is supposed to cure. In other words, what is the problem we are trying to solve? I know the veterinarians and the chemists feel that the 48-hour rule puts an unfair burden on them. The rule was never designed to put an unfair burdenon any one individual or any one group. But the racing commissions throughout the United States, where parimutuel wagering is legalized, have the very heavy burden of enforcing the various state laws which legalize wagering.

In discussing this rule, it is proper that I point out to you some of the problems which confront our commissions. Those problems are entirely different from those which confront you as professional people.

Racing originally began—and no one here will be more qualified to recognize this, I think, than Mr. Marshall Cassidy who is a long-time Steward of the Jockey Club—and still is a sport in which everyone in it is dedicated to improvement of the breed. There was a time when racing did not involve gambling or wagering. Then, as racing grew and as more and more tracks came into being, and as the purses grew from $10,000 to $15,000 to $50,000 to $100,000 to $200,000, and maybe some day half a million, more people went into racing; more people spent more money on horses, and when they ran and had a chance to win more money, they gave more thought as to what they could do to insure their winning. That is one of the curses which we have today. It is that there are not enough sound horses. In the minds of many of us we feel there are horses that are running back too often, and being flown from one big stake to another, to the end that you almost never encounter a normal, sound, racing animal.

Those of us who have owned race horses recognize that a slow horse doesn't get sore very often; it is the good one that is sore. He is the one that is the problem to the veterinarian who is taking care of him. But I want to point out to you the tremendous position which racing enjoys in the United States. It is the No. 1 spectator sport. It handles the most money, and it is one of the most colorful sports in our nation. It could not possibly enjoy that position if it did not also enjoy the complete confidence of the wagering public.

It is not too long ago that the National Association had a real fight on its hands to legalize the urine test. As you know, in the last twenty-five years we have made tremendous strides in developing many things which insure that the wagering public gets a fair shake for its wager, and that all of the facts relating to the condition of an animal are made available to each and every person inclined to wager.

It is the Racing Commission's responsibility to see to it that a person putting $2 through the wicket at the mutuel window has all the information that is available about the conditions of the

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race, the conditions of the track and the conditions of each horse entered in the race.

To that end, over the years, we—and by "we" I mean those of us interested in racing at all levels and in all segments—have developed the electric starting gate. We have developed the photo-finish, a photograph which establishes beyond any question which horse won, which was second. We have developed the photo patrol which now takes a photographic recording of every step of the race so it could be re-run, to determine whether a foul was made. The totalizator people have developed the art of making the odds on each animal available within a matter of seconds to the public.

All of those things point toward building confidence in the minds of the racing and wagering public. We go beyond that. We even see to it that every time a horse runs, the track timer, or the official timer, records the work of the horse.

Mr. Sam Perlman, who owns Racing Form and is a staunch supporter of clean racing and represents the official publication for the National Association, is militant about seeing to it that the information given to the wagering public is accurate.

Now, being aware of all of those things, we in racing are very, very concerned about permitting any one individual to have the right to do anything which might alter the racing form of that animal. I am not saying that it is wrong to give an ailing horse something that will help him. I do say that it is wrong to let a trainer or a veterinarian or a layman have the right to determine whether it shall be given or not, to the extent that it affects the horse's performance, and there is a big difference. Because the person wagering assumes that the animal is a healthy animal and that the animal is qualified to start, and that the animal will run consistently to his or her former form.

If that form can be altered at the will or pleasure of any individual or any entity, then the information which the public has is not completely factual. I have tried to say that is the problem. Whether the 48-hour rule solves it or not, I cannot completely say. But it was with that thought in mind that we established a 48-hour rule.

I think I want to reiterate again exactly what Mr. Inglis said, and that is this: The 48-hour rule was never intended to deal with stimulation, as such. That is adequately covered in the corrupt practices section of the rules of the respective states, in that, in essence, they say it is never legal to give a drug to a horse which will affect its racing form.

The 48-hour rule was essentially created to be a medication rule, to prevent the doctoring of horses, literally, to the starting gate, and to that extent we feel it is a worthwhile rule.

In closing, I want to say, too, that I have an open mind on this subject. I do not think it is a perfect rule, and I am delighted

that a group such as yours, so close to racing, recognizes the problem and is shouldering the responsibility of coming up with an intelligent solution, with us.

Thank you for hearing me. [Applause]

MODERATOR WOODCOCK: Thank you very much, Commissioner Bruno.

Now I would like very much to call on Mr. Manfuso who will give the views that his organization, the H.B.P.A. has set forth recently. Mr. Manfuso!

MR. JOHN MANFUSO: Mr. Chairman, Members of the American Association of Equine Practitioners: Usually at these conventions the representative who has been invited to address you uses the opportunity to voice some particular grudge or beef. I want you to know that the organization I represent, the Horsemen's Benevolent and Protective Association, has no complaint, no beef with your fine Association.

Of course, as horsemen, owners and trainers, we have the physical problem of the horse still with us, starting with shelly feet, fractured os pedis bones, contracted heels, navicular trouble and ringbone, osselets and sesamoids, suspensories and fractured splint bones, bowed tendons, puffed and hot knees, and on through the anatomy of the horse.

We should categorize these problems against inadequate basic research and not charge them to the veterinary profession. There might be one small problem, however, that is a cause of complaint.

When I spoke to you last year I discussed the subjects of stimulation and the 48-hour medication rule, and offered our cooperation in your endeavors to establish a program and fund of perennial income for basic research in the horse. Those subjects still seem to be the most acute for mutual consideration.

We all know that there are cases of undetected stimulation occurring, even though the percentage is relatively small. The H.B.P.A. has gone on record and is unalterably opposed to any medication for stimulation which will artificially improve the running ability of a horse beyond his normal capacity. We are certain that your Association is of a similar mind. However, I have heard the argument that some veterinarians feel that if they don't do the job, the trainer will do it anyway, so why not pick up the fee? I can't go for it, gentlemen, no more than I could go for anyone who would sponsor a boxing match and have one contestant with brass knuckles. It is absolutely contrary to any principle of fair play and sportsmanship. To me, it makes one almost an accomplice of moral thievery. I am certain you agree with me.

The 48-hour rule, the 48-hour medication rule, is more acute today, much more as many of you know, than it was last year. We definitely need your help on this rule. There is a wide divergence of opinion in our own organization.

I was handed a letter from the Florida Division of H.B.P.A.

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which has voted unanimously against this rule. I know that the New England Division has not only voted unanimously against the rule but has also polled the entire national body of H.B.P.A. for a decision on the matter at its coming convention in California in January. At present our association nationally is on record as approving the rule. Our division in California has approved the rule. But there is a tremendous difficulty involved, gentlemen, with horsemen themselves. They cannot understand the need of a medication rule, especially since stimulation is not involved. Since the malpractice rule will cover stimulation, irrespective of whether a drug is administered 24, 48, 72 or even 240 hours prior to the race, they cannot see any necessity for the rule, other than maybe the necessity of requiring standard performances or standard finishing of a horse, and they question whether this should be obtained through the control of some proper medication, rather than I think, as Mr. Cassidy has indicated to you, by the action of the officials in control of racing to question, to find out why a horse does not perform consistently in his chart performances.

To my knowledge, there has been some improvement in your quest for a basic fund or a basic research program, but to this point there has not been any definite or financed program, you might say, crystallized.

Our opinion is that the money is available for this program but it has to be a necessary and specific one. I am very much afraid that the membership of your Association and your officers are going to have to lead the way for the racing industry.

I want to thank you for inviting our association to be present and speak at your 1959 Convention. I also want to compliment you on the acceptance you have received in the racing industry, and especially to congratulate you on the quality you have received from your officers who have represented you.

At the recent New Orleans Convention of the T.R.A., the address of General Kester and his remarks from the floor on encephalomyelitis, in my opinion, created a profound impression. I doubt whether nine out of ten, of the large audience present, knew that equine encephalomyelitis was, to a degree, a misnomer, that the disease was actually a disease of birds transmitted by mosquitoes to other animals, and that there was no record of the disease being transmitted from the horse to either humans or any other form of animal life.

If this is typical of your Association, gentlemen, you are destined to make a tremendous contribution to the industry of the running thoroughbred horse. [Applause]

MODERATOR WOODCOCK: Thank you, Mr. Manfuso, and thank you, also, for digressing a moment. We are very proud of the work that General Kester has done for this organization.

I would like to call now on Dr. Oester who represents the National Association of Racing Chemists. [Applause]

DR. Y. T. OESTER: Mr. Chairman and Members of the Equine Practitioners: I would like to agree with several points made by the previous speakers, particularly the first one, I think, made by Mr. Cassidy, that the rule should not be called a stimulation rule; it probably should be called a drugging rule, or some other appropriate phrase. We certainly have to consider, I think, the widely accepted fact that depressive drugs also should be listed as a reason and cause for taking action, when they are used illegally.

I would like also to ask and second the question he asked, why should one drug, phenothiazine, be taken out of the whole list of drugs we have, by one commission and be barred for two weeks? I have had no communications from California about this and, therefore, also wonder how this came about.

I would like to second the remarks of the last speaker in regard to basic research. There is no question that many of the points, let's say differences, between people who are speaking today and who will speak together in groups around here today, could be ironed out if we had enough information about the action of drugs and about what they do to horses. We still do not have adequate knowledge in this field.

I think my purpose, my primary purpose, would be to bring to your attention a number of basic facts about drugs and their handling by various animals, including the horse, so that we could then use this as a basis for possible discussion and elaboration of the various aspects of the rules we will be discussing.

I am also aware of the fact that the biochemical knowledge or the pharmacological knowledge about a drug is not enough of a basis by itself on which to make a rule or change a rule. Mr. Bruno's remarks certainly brought that home clearly, that there is no way we can avoid, particularly, considering the aspects of wagering and the aspects of the public in regard to drugging or not drugging a particular animal.

The first general consideration I would like to make is this, and I will ask it in the form of a question: Should any horse ever run under drugs of any kind? To repeat: Should any horse ever run under any drugs of any kind or at any time? My own feeling, and I think that of most of the chemists in our group, is this: Any drug can or may affect the performance of a horse. That means *any* horse. We would not like to point at the drug and say it is a stimulant or depressant. Almost any drug, it can be shown in proper animal experiments, will have other effects than the ones you may be familiar with and for which it is used.

So that I think in any basic consideration of rules and regulations we must remember this fact, that any drug can have an effect on any horse. Some of the drugs do this directly by their action on blood pressure, heart rate, and so on. Other drugs may do this in an indirect manner, where the individual horse or in-

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dividual animal may be hypersensitive to the drug or react in an abnormal manner. Or, in the case of overdoses, new effects of the drugs may be brought into play which are not present in routine dosages which are used. As an example, of this indirect action, such drugs as aspirin, acetylsalicylic acid, and some of the other pain-relievers, like pyramidon, antipyrine, these are given primarily to relieve a symptom in the horse. Or, in the case of the human, to relieve a pain, either headache, or ankle or knee, whatever the case may be. But a symptom really represents some abnormal tissue change in this animal. In other words, there was a pathological change in the animal which would produce this symptom. If our drug actually eliminated or made away with the underlying pathology or underlying basic tissue change, we would be treating the disease directly. As it is, almost invariably we are treating not the primary disease; we are treating a symptom, or one manifestation of the pathology.

It is in this area that I think we must agree that the use of these agents will change the performance or the activity of this animal because, as soon as we relieve pain, we have changed his performance. That does not mean we have eliminated the underlying cause, the pathology in the tissues. It means we have more or less masked it and, obviously, the underlying change may still be present, or may be present in a more aggrieved form after our treatment.

Undoubtedly, I think, the animal running with pathology present, or abnormal tissues, or tissue reaction present, certainly should not be on the race track.

This type of analysis which I have given for pain relievers can be given for almost any drug that is used. This leads me to an answer to this question I have asked. I would say that no drug should be given to any horse before racing. This is because there is a basic pharmacological fact we can't avoid, namely that there is going to be some influence in this animal by any drug that we may use.

A second basic consideration leads us to the 48-hour rule. What happens to drugs when they are given to animals, particularly to the horse? Are they eliminated in the course of 48 hours, if given at the very early time in the 48-hour period?

Therefore, the question would be, is this 48-hour rule valid from the biochemical or pharmacological standpoint, not from the standpoint of, is it a good rule, or good control of racing in general.

Not all dugs, if given 48 hours before a race, will be eliminated from the animal, or secretions from the animal, within the 48-hour period. We have had a number of examples of this. One of the most common is that of procaine penicillin given two or three days before a race. It will still show up as being a present in the excretion of this animal. Certain other drugs are not all excreted

within the 48-hour period. If I had to make an estimate or guess—I do not have any exact figures—I would say probably 85 to 90 per cent of drugs would usually be eliminated, if given in a regular or routine manner, in the space of 48 hours.

However, there are many other possibilities available, with the advances being made in pharmaceutical preparations. We can give drugs by mouth under the so-called time disintegration factor or time disintegration method—"time disintegration" means this drug will not take its effect immediately, or within one or two or three hours, but take a number of hours, usually from four, eight or twenty-four hours before the drug casings are broken down, and the drug is released for action.

This obviously opens up almost unlimited vistas of drug administration. If you can time, let us say, the release of the drug in the gastrointestinal tract, if you can time the release properly, you can get by the 48-hour rule, of course.

Another general preparation now commonly available is a depot preparation. In this situation, the drug is given in a muscle area usually, or subcutaneously, underneath the skin. It is not immediately absorbed here but is very, very slowly absorbed in the course of two, three or five days, as the case may be. Again, this is obviously an area which the 48-hour rule does not cover, even though the administration may be for medicinal purposes or therapeututic purposes.

So in answer to the question, is the 48-hour rule valid, I must say it will not hold for all drugs. As I have already mentioned, it may hold for a number of drugs, if given in routine, ordinary ways, but will not hold by the time disintegration method, or by depot preparations.

I think those are the major items I wanted to bring to your attention. I have not answered, I am sure, the question of whether legitimate therapeutic use can be distinguished from illegal use of drugs. This really is a tough one, I am sure, for all of us to answer. This is a question we must face, upon which I cannot give you any answer or any real data.

I also would like to say, as a final extension of these remarks, that there is no way for the chemist to determine how much drug was given to a horse, or when it was given, if he is confronted with a one-sample analysis, such as he ordinarily is, taken at a random time after the administration of the drug. From this random sample, there is no way we can tell you how much drug was given, and at what time it was given. We can sometimes come up with some fairly good estimate. In this sense, we can say in our experience we have never recovered this amount of drug two days after it was administered, or three days, or five days, whatever the lift may be. We cannot give you any close timing. This all depends upon a number of factors which we will not have time to discuss here.

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In conclusion, I would say we have, as the Association of Official Racing Chemists, no hard and fast decisions about how this rule against drugging and doping should be written, but it does seem we should base our preliminary thinking and our discussions on these various facts we have discussed here. I think, with them, we will be able to come to a fruitful, final resolution of this rule.

Thank you. [Applause]

MODERATOR WOODCOCK: Thank you very much, Dr. Oester.

Now I would like to call on Dr. Reed who, as you all know, is a practitioner in New York and Florida, and has operated under both rules, for his impressions and his thoughts after hearing what was said here before him. [Applause]

DR. WILLIAM O. REED: Gentlemen, this is the first time that we as veterinarians collectively have had an opportunity to discuss this problem in the presence of racing officials.

To my knowledge, there has been no attempt made by any of these officials, and I am not saying this with any disparagement toward them—with the exception of Mr. Cassidy, I have been most fortunate to be under his jurisdiction for a number of years, and I can't tell you how fair and how rewarding it is to work under him. But we, as a whole, have never had the opportunity to sit down and discuss with them our problems, and why we have these problems, and what they are. I don't think these gentlemen realize what the situation is. I quite understand their problem and I am sympathetic with it.

First of all, we have the basic purpose of the stimulation rule, or whatever you want to call it, which was to overcome stimulation with narcotics. That was the No. 1 thing, originally, narcotics and, of course, local anesthetics. I think we all agree that that is a "must" to eliminate. I don't think it is fair, or it is ethical, or it is humane or anything else to use any of those medications for that purpose. The practitioner has many problems with this rule that do not reach the surface. First of all, we still agree, and all agree, the public should be protected. They are the ones who are most affected if a horse is stimulated, or some particular owner might be, but the public should be protected at all times. It is something that no one mentions. What about the owner of that horse? Nobody says a word about him. He may pay $60,000 or $70,000 for a horse. All of a sudden that horse is a bleeder. What is he going to do with him, shoot him? He can't run him at a track where the competition is less and expect him not to bleed. He is going to bleed any place. He will bleed when he works. I have seen horses bleed galloping. Jockeys must be protected, too. I think they risk their lives daily. I think they should be permitted the privilege of getting on a sound horse. There are borderline cases, which even veterinarians examining horses at the race track

will admit. You talk with them and they will say, "This horse can run this time but I'm not real sure, I don't know how he will be the next time." I take my hat off to those fellows. In New York we have some real good ones. That does not preclude a jockey from riding a sore horse. There have been accidents with jockeys getting on sore horses.

Because of the ramifications of the 48-hour rule in different sections of the country, I am going to take the privilege of calling on certain veterinarians at random, veterinarians who are in different sections of the country, and ask them, from the floor at this time, to give their impression of this particular situation and why and how it affects them.

First of all I would like to call on Dr. Joe Burch from New England and Florida.

DR. JOE E. BURCH [Miami Shores, Fla.]: I would like to say, first of all, that there have been a number of comments that are direct contradictions of opinion or exact opposite beliefs. As veterinarians, we see this problem daily. I think in the Executive Board last night the majority felt that the 48-hour rule is a bad rule because it limits the practitioner's facilities and know-how of helping his client. Our basic concern is the horse. There are a lot of horses we treat that need medication sooner than the so-called 48 hours. Dr. Oester asked the question a few minutes ago, "Should any horse ever run under the influence of any drug at any time?" His answer is "No." I think most veterinarians feel that the answer should be "Yes." For the same abnormal tissue change that he speaks of, we use anti-inflammatory agents. We do this as a protection to the investment of the owner.

The anti-inflammatory action you are going to produce with certain of these drugs allows the animal to be raceable. Many of those inflammatory changes are not correctable with long-term rest or therapy that we have at the present time. It is very true that some of them exert a masking operation. The masking operation does a job on the horse, allows him to be raceable.

Butazolidin reverses the inflammation of an ankle, of a knee or any of a number of conditions that Mr. Manfuso spoke about adequately, so that a horse is raceable. It does not represent a capital investment going to zero, on the part of the owner. We come into this picture because we are consulted daily: What approach should we take, or what can we do, as Mr. Bruno said, something to insure winning. What about something to insure losing? Many times you are treating a horse, not in an attempt to increase his speed or value, but to reverse that procedure and keep him from devaluating a $10,000 horse which develops a certain type of lameness. We have at our facility now steroids, cortisones that will reverse inflammation and allow the horse to be raced, with no danger involved to the jockey or the public.

Many of us feel that the public would be deceived if you did

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not do something to reverse his inflammatory process and assure an adequate performance by the animal.

So that this thing works two ways. I might say then, along that same line, this change that has come about in the rules has been reversed from a rule that covered stimulants, depressants and local anesthetics, to become a rule now that embraces all medication, and most veterinarians feel there is a vast difference.

Bleeders were mentioned, and rightfully so. There are many other problems in lameness that we have, that fall into the same category as a bleeder. I think that is enough for me.

DR. REED: Thank you.

Dr. Copelan from Chicago and Florida, I would like to have your impressions of this.

DR. ROBERT W. COPELAN: Gentlemen, I have had an opportunity to practice under different types of this rule. I practiced under the 24-hour rule, the 48-hour rule, and no rule at all in the state of Kentucky. I don't exactly mean no rule at all, but I practiced under a very fair rule as is practiced in New York, in my opinion. I have similar emotions about some of these things.

First of all, I think of human life, in the case of the rider, in the case of a very simple disease in young horses that we see a lot of extreme muscular soreness. I know a lot of men present realize that horses plagued with this condition are dangerous, even at a gait as slow as a gallop. This condition can be reversed with the use of steroids and phenylbutazone and some other drugs, and can make this horse, in my opinion, very safe to ride.

Bleeders I feel should be able to be treated. We all know of cases of very expensive horses that are bleeders, not being able to mention by name.

Without treating these horses, some of the managements and racing secretaries at different tracks would be without the services of these horses to present an attraction to the wagering public to come to the race track and bet on the races. People are actually interested in better horses. That is what I think makes better racing.

I think that the 8-hour rule is not a good one. I am not sure I can come up with a substitute for it that is exactly perfect, but I know that practicing veterinary medicine, with the education that I received in a proficient veterinary college is quite difficult to do under the 8-hour rule or under the 24-hour rule, or under any hour rule.

My armamentarium is considerably depleted by not being able to use the drugs that I have been taught to use for various conditions. I do not feel that I can do a service to the owner or the trainer without the use of these drugs, the way he expects me to do this. When he calls for a veterinarian, he wants somebody who is proficient and qualified to be able to treat these

horses, as Bill says, for the man who puts on the show should be entitled to this service.

DR. REED: Thank you, Dr. Copelan.

Dr. Trussell from Charlestown, West Virginia, I would like to have you express your opinion.

DR. WILLIAM E. TRUSSELL [Charlestown, W. Va.]: As far as the 48-hour rule of medication, I practice with and without it. I have noted over a period of time that, as far as the percentage of winning favorites, there is very slight variation. I realize probably the question is somewhat debatable.

We as veterinarians I think are charged with the problem of keeping horses sound and normal, and not to artificially stimulate the horses. I am definitely of the opinion that the specific handicapped horse is entitled to professional aid, providing this aid is under professional supervision. I think it will definitely make the horse perform nearer his normal ability.

If I may digress just a moment or two from horses to human beings, athletes and so on, there isn't any question about it that they are often medicated. I did have the experience yesterday afternoon, of seeing a professional football game. I don't think there is any question about it that one of the players had a nose hemorrhage. They brought him to the side of the field; the doctor worked on him, and fifteen or twenty minutes later he returned to the ballfield and played ball, and went along all right.

If I might go back further, personally, approximately 1940 the medical authorities gave me up as an arthritis victim. I do have ankylosis of the vertebrae in my spine, but I miss very few days of work. I did receive quite a bit of medication, and I am very thankful that I did, and that I have been able to go along with my profession over a period of years.

DR. REED: Thank you, Dr. Trussell.

Dr. Ed Lang from Louisville, Kentucky.

DR. EDWARD M. LANG, JR. [Louisville, Ky.]: Gentlemen, of course in Kentucky we do not have any law, but to me the main thing about this 48-hour law is due to the great interest in racing, and the economic value and what revenue is derived out of it. Horses are costing more money. There is an ever-increasing number of horses each year at all tracks. With the 48-hour law, as I see it, the hardest man hit is the trainer. How can he tell, with the increasing number of horses, when he can get a horse in? With the 48-hour law, there is no way that he can train the horse properly. Therefore, that can lead to the damage of the horse, that would not be fair to the public either, without letting veterinarians administer what they see fit as trained professional men.

DR. REED: Thank you.

Gentlemen, in conclusion, I have one little statement I would like to make and that is this: I feel that any horse in training is an athlete. I think he should be considered as an athlete and be

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given every opportunity to perform to his normal ability.

It is not unknown that one of the greatest tennis players we ever had in this country was a diabetic. He was given insulin constantly, shortly before he played. That is just a simple example.

It is my feeling that sooner or later these rules will have to be altered to fit our present situation. It may take time, and we may have to do a lot of work.

We are most happy that we have been called on at this time to express our opinion. Thank you very much. [Applause]

MODERATOR WOODCOCK: Thank you, Dr. Lang, and gentlemen from the floor.

I think we have had a few very important points. I would like now to call on Dr. Teigland, a practitioner from Florida.

DR. M. B. TEIGLAND: I am honored to represent this group as a member of the Equine Practitioners, and to the opposing members on this panel, as to my views, I hope I may present myself with the humility demanded by Arthur Godfrey, but, on the other hand, I hope to express the convictions as I feel them.

We have all discussed the purpose of the original racing rule regarding narcotics, and I think this is all well understood. The purpose of my practice on the race track, in general, is to maintain the best performance of the race horse.

I really believe that our major problem with horses might be classified into two groups. I think one of the most important problems in treating the horse is the treatment of inflammation. This covers many types of inflammation, whether produced by infection, by trauma, or various and sundry accidents that occur to the horse.

We also are faced with the problem of treating deficiencies in the horse. I do not refer to the horse, the deficient horse; I refer to the horse who is not doing well. We make every effort to determine why this horse is not doing well, and we make every effort to correct this deficiency.

I see no reason, in my own personal opinion, why it makes any difference whether this deficiency need be corrected over the period of a month before he races, if it is possible to do so, or whether this deficiency is one of such nature that it must be corrected within a reasonable time before he runs.

The problem of the pathology of the horse was mentioned by Dr. Oester, a member of the panel. I think his interpretation of treating the pathology of the horse infers we do not relieve the pathology of the horse. I do not believe this is quite correct. I do not mean to differ with him, but it has been my experience that I have had a number of inflammatory processes. I do not want to single out one drug, as I would not want to tip you fellows off as to what all I do know. [Laughter] But let's do talk about a couple of them.

If you have a horse who is lame in the left ankle, the left

ankle is accessible with the hypodermic needle. There are anti-inflammatory compounds, and, if you inject one of them, he stays sound, as far as we are concerned, forever, at least for our racing season. Maybe, in many cases, the reason this horse has to be injected, let us say, every week or every two weeks, or whatever it is, is due to the ability of this joint to maintain the therapeutic amount of the anti-inflammatory compound we use. Then we are correcting a condition of the horse, and we are treating this horse. I do not feel that there is any difference, that treating a horse to correct a lameness in an ankle that is accessible is any different than treating a horse the day before he runs because he has an inflammation which is inaccessible to treat, or probably not even within our ability to diagnose the condition.

However, if we can prove through medicating this horse in training that it corrects the condition, this horse is obviously trained with this medication. This horse is recorded in the Racing Form as to his work with this medication. No trainer in his right mind, and no veterinarian is going to allow a sore horse to train, and take the chance of this horse being out of coordination and fracturing sesamoid bones, fracturing any particular part, or bowing a tendon. Consequently, horses are being trained under medication.

We are responsible, in my opinion, to treat this horse for the purpose of racing. That gives the true form of the horse. It was mentioned that a horse pulls up sore and the public knows this. So, the next time this horse comes to the post, the racing public is not aware that this horse is to be treated. Consequently, they do not figure this horse has a chance, and he jumps to 20 to 1. Eighty per cent more horses pull up sore unknown to the racing public. If they are even money shots, they go back to the post as an even money shot and, consequently, are expected to perform the same way. Without medication, they cannot perform the same way, and the racing public is being fooled.

They speak of exceptions to the rule as to treating bleeders. There, again, I see no difference between making an exception to the rule to treat a bleeder than an exception to the rule to treat any inflammation. One is as serious a problem to the racing public, to the stewards, to the jockeys, whoever is concerned, as is the other.

Consequently, if we are going to make exceptions, we have a cumbersome task on the part of racing stewards, on the part of veterinarians, on the part of everybody involved in racing. The better solution is to allow veterinarians to medicate horses and, I hope, in a professional manner, and allow these horses to run according to their form.

I would like to make a comparison as to the two basic problems that we are discussing: medication versus stimulation.

Let us talk about stimulating a horse, and let us point out

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some of the major people concerned with racing. I will try to cover them all. First of all, the official does not want a horse stimulated. The jockey surely doesn't want the horse stimulated. The trainers, in 99 per cent of the cases, I truthfully believe, do not want the horse stimulated. If it weren't 99 per cent of them did not want the horse stimulated, 90 per cent of them do not want the consequences. The owner, in most cases, surely does not want the horse stimulated. The race track association, the betting public and, surely, the veterinarians do not want to operate in this manner.

As to medication that is beneficial to the horse—and I am talking about medication of the horse—I would like for anybody on this panel, or anybody from the floor to name the medication that will allow the horse to run better than he is capable of running. I do not think there is any such medication. But there are many that will allow him to run as well or nearly as well as he is capable.

The official likes to see a nice clean day of racing. It is always a blemish to a racing card to have a couple of horses pull up sore. The jockey risks his life in riding a sore horse and, unfortunately, has to get on an occasional sore horse that could have been corrected, from the soreness. The trainer has the responsibility to his owner and somewhat to his family in making a living, and he wants the horse to perform at his peak performance. Surely the betting public wants the horse to perform to the best of his performance, and you and I, as veterinarians, want them to do so. I would like to say I don't consider me part of the betting public.

If these medications were as exceptionally earth-moving as some people infer, I wouldn't make my living treating horses; I would be part of the betting public. [Laughter] Nobody benefits from the horse that is stimulated. Everybody benefits from the horse that is properly medicated.

I want to take my hat off to our Canadian friends, when they put on a stake race, they put on a stake race. I witnessed the Queen's Plate in Toronto. My wife and I attended. The Queen of England was present and entered the paddock on two occasions, between races. Recently I have seen comments that this is the sport of kings. Let's keep it the sport of kings and let's eliminate the bums.

I am sorry George Badame is not here because I am going to quote him as to his conversation. Maybe George is here. George, I am going to use your statements.

The Queen of England came to George Badame, and I noticed she spent more time talking to George than she did to anybody during the paddock interim. Her main problem to George was, "What do we do with sore horses?" The sport of kings goes on, men!

There is another little problem that probably should not

involve this particular discussion because it has already been discussed and should not be allowed, but you are opening the door to the unethical druggist, to the unethical trainer, to anybody in the game who is unethical. Unfortunately, we have these people. The 48-hour rule protects these people in a nice, cunning manner. They have no ethics.

We as veterinarians operate under the 48-hour rule, and I have operated under both, and I feel seriously handicapped under the 48-hour rule. It is obvious that, if I do not medicate the horse, the unethical druggist will rear his ugly head and, men, this is when we get into difficulty as to proper medication of horses.

Uniformity of rule obviously is what we are all trying to strive for, and I do not infer that I am the guy who knows the answer to this question. I resent the 48-hour rule, with its inference.

I had occasion to speak to a group in which there was a judge. I asked the judge, "Sir, would you like to have a rule posted that no judge sits on the bench within 48 hours after having a drink? What is the inference, Judge?"

He said, "It is an inference that we are all drunkards."

This same inference applies when you have 48-hour medication rules posted for veterinarians. The big problem about uniformity of rules, horses come to our area where they have been racing under various rules and they, of course, have received various medications. These horses come from New York. They are performing splendidly. They may be the horse of the year; they may be a $3,500 plater. I don't think we should eliminate either. We need them both. But they come from New York, come from another race track to our area where they have the 48-hour rule.

If the horse was run under medication in New York, how can he perform according to form in Florida? However, I am sure there is uniformity of agreement that there should be a uniform rule, and I hope that this group and this panel will consider the uniform rule that we are going to propose.

Thank you,

MODERATOR WOODCOCK: Thank you very much, Dr. Teigland.

DR. JAMES L. TEMPLE [Arcadia, Calif.]: It has been my opinion with butazolidin, if it is a particularly early case, one that has not been repeated and the ailment not too long, the lasting effect is greater than 48 hours. It will last him three to five days.

I would like to bring up two other points. In California no one knows forty-eight hours ahead of time who is going to race and who is not. They come out less than 24 hours and closer to 18 hours. So there is no way of knowing that a horse you treat today is going to be in within 48 hours. That is a discrepancy.

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DR. E. M. LANG [Louisville, Ky.]: Talking about butazolidin, is there really a test? Is there a test the chemists can specify?

DR. OESTER: The drug has not been in our regular testing program for the last several years. Within the last year the Colorado group of chemists have devised an efficient testing procedure which is now available to all chemists in the United States, or in our association. They have run through animals where they have given the drug.

DR. LANG: In the states where there is no 48-hour law, are you going to be naive enough to tell me that nobody has used it, and the chemists are not picking it up?

DR. OESTER: After listening to the talk, I would not say no one has used it. I mentioned this test being available within the last year. This has been widely available to all chemists. Before this time it was not widely available. I think most chemists were not considering the drug to be tested for.

DR. LANG: How is it that more is picked up? The only place they picked it up was in New Mexico, and the case in Michigan.

DR. OESTER: This would rest entirely with the local chemists. I cannot speak for them at all.

DR. LANG: Do they test the same as you test?

DR. OESTER: I assume they do. I have not been in their laboratories to find out if they do. The only way I can be sure every other laboratory in the country is testing the same is that we get the same bulletins; we exchange the same type of information, one with the other. So, all information I have is available to other chemists in the United States. I cannot say they are or are not testing the same as I do here.

DR. EDWARD M. UMANS [Oak Park, Ill.]: As far as history is concerned, with every disease we have ever had, we have come up with some kind of solution, a drug, vaccine, or something of that nature. We have just about licked polio.

Now we are coming into an area of new drugs that we have all used, and used successfully. We know how they work. The racing laws are antiquated and they should come up to the newer forms, as the medical profession has come along. They, too, should kind of cooperate and go along with the advances in medicine.

MODERATOR WOODCOCK: That is an excellent thought, Doctor. The reason we are using this preparation, butazolidin, at the moment is to stimulate this kind of thought for the future. After butazolidin something else will come along.

DR. FRITZ A. HOWARD [Warrenton, Va.]: Dr. Reed spoke about the owner's investment in the horse. He pays $60,000 or $200,000 for the horse. The horse comes up lame. It may be bursitis. The use of butazolidin or steroid could correct the situation.

I think the chemists have to classify the type of drugs being used, put them down as stimulants, steroids, or other things, to relieve the inflammatory conditions. I have calls coming in. They say, "The 48-hour rule is in force and I can't use butazolidin. How about taking a little white tissue out for me?"

I know one horse that has been raced in the East, at the big tracks, has been nerved from the knee on down. I would rather see a steroid or butazolidin or some other drug used to relieve the inflammatory condition than to have the white tissue taken out. It would be safer for the public and safer for the jockey, and the whole way around. But your chemists or your racing officials are definitely going to have to classify what type of drugs you can use to relieve that, or what type of drug is a stimulant.

MODERATOR WOODCOCK: Do any of you have any comments you would like to make?

MR. INGLIS: I would like to ask one question, Dr. Woodcock, and I can address it to you. Suppose, for example, this past season in New York your chemists had come up with a positive butazolidin, would the New York Racing Commission have considered that grounds for disciplinary action or not?

MODERATOR WOODCOCK: According to our rule in New York, that is an entirely a discretionary measure in the hands of the stewards. I do not know what the stewards would have decided. I believe this, that if sufficient evidence were shown that an overt act had been committed on the part of the trainer or the owner or anybody involved, yes, a suspension, and disciplinary action and reference. The disciplinary action would depend upon the severity of the crime, we might say.

Mr. Cassidy, would you comment on that?

MR. CASSIDY: In New York, if anything is found in the saliva or the urine, the stewards investigate the case completely, including whether the owner or trainer had any prior knowledge that the horse was going to run a better race and that the betting indicated they were taking advantage of it. That would have quite an effect on what they would do with the case. But unless they found, to their satisfaction or complete satisfaction, that an attempt was made to defraud the public, I doubt they would take action as a stimulation case, or violation of that particular rule.

MR. INGLIS: I would like to ask just one more question. There has been a lot of talk here about protecting the owner of a horse by medicating a horse to bring him up to his true worth. I would just like to throw out the question for consideration: You have two horses that have equal potential. They go into a race. Everything is equal, including the riders and all the other factors, but one horse is sound through his natural condition and the other is sound through butazolidin. Which horse actually deserves to win the race?

Shouldn't we also consider the investment and the value of

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that naturally sound horse in which some other owner has a big investment, too?

DR. TEIGLAND: I would like to comment on Mr. Inglis' question. If this is true that we must protect the man who has the sound horse, let's change the rules of racing. This isn't my problem. Let's have conditions of racing for sound horses and let's have conditions for unsound horses.

MR. BRUNO: I would like to invite comment on two points. First, I think it is important, gentlemen, that you recognize that we are not necessarily quarreling with what you as professional veterinarians are doing. You must remember that, when we write a rule, it bears not only on you but it bears on every licensee. I am sure that there is more injecting and medicating being done by trainers, owners and laymen than there is by veterinarians.

If we were dealing strictly with veterinarians, I think our problem would be 90 per cent solved, because the veterinarian could, under a formula, report to the state veterinarian what he is doing, when he is doing it, why he is doing it and how he is doing it, and our track veterinarian would recognize the validity of what he is doing and advise the commission as to the appropriateness or inappropriateness of what is being done. But, when we set out a rule, then every person who has a license comes under that rule. At the moment there is no rule which bars a trainer from injecting a horse or giving a drug.

The other question I would like to ask, and Dr. Burch put it very clearly, I think, and that is this: I think it is agreed that butazolidin helps a sore horse. If the horse is helped today with butazolidin, and the horse runs a week from now without it, shouldn't the wagering public be told that? Shouldn't we say, "This horse is going today with butazolidin. A week from now he goes without it." If you don't, the public is being misinformed, or ill-informed.

DR. REED: In answer to that first statement, I think if the hypodermic law were to be enforced, as it should be, by officials, not veterinarians—veterinarians cannot go around rooms and enforce it—that would eliminate the trainer from giving that medication. You are stating that the trainer is the one you are worried about. All you have to do is enforce that, and it is all set.

MODERATOR WOODCOCK: Care to elaborate?

MR. CASSIDY: We have a rule in New York which adequately covers that. It says that no person shall have in his possession in or about any race track or shall use appliances, electrical, mechanical or otherwise, rather than the ordinary equipment, of such nature as could affect the speed or racing condition of a horse. That is part of the rule that has to do with stimulation, but there is another one that says that the stewards or the officials have the right to enter the grounds or the rooms or the possessions of any horseman and, if they find a hypodermic needle or any instru-

ment to inject a horse, that man shall be dealt with by the stewards.

DR. H. M. SMITH [Hanover, N. J.]: Just a second thought. Mr. Bruno seemed quite concerned about the protection of the betting public, as far as the form of the horse as it is published in the Racing Form and racing programs. We have no way of knowing whether that horse is in the race because it needs an extra race to come up to form, whether it is in there really trying, or just giving it another race. I think that is something that there is no way of controlling, as far as protecting the betting public.

MR. BRUNO: Of course, you can understand that no individual could say every trainer who enters a horse is trying to win but I can tell you what the state laws say,, almost without exception, that every horse entered is entered to win. If anyone is caught entering a horse without the intention of trying to win, we would penalize him, as quickly as we could call a commission meeting.

DR. E. M. LANG [Louisville, Ky.]: What about the horses used to fill a race?

MR. BRUNO: They don't come in until they have a chance to win.

DR. ROBERT H. BAKER [Montclair, Calif.]: On the Commissioner's question as to whether a drug would be used consistently on certain horses, I think the value of the purses in present-day racing will cause most trainers to continue using a successful drug. Also, there are far easier ways of using drugs than not to give butazolidin.

DR. C. H. BURGER [Bakersfield, Calif.]: I think this 48-hour rule encourages the trainer to inject his own horses. He realizes the veterinarian is in no position to inject the horse within the 48-hour period. Therefore, he goes ahead and buys steroids and B_{12} from some unethical druggist, or some other source, and injects the horse himself the same day. I think this is detrimental.

DR. OESTER: Since pathology was questioned or talked about a little since I sat down, I would like to return to that briefly, not with the idea of confusing us but for clarification.

There have been about half a dozen case histories of butazolidin brought up by different ones. It seems to me each one sort of stressed the fact there is continuing pathology in horses that is not cured with butazolidin. The horse went off the drug and it did not perform as well as it did when it was on, because it went back to his previous pathological state. We may be suppressing it, we may be masking it, but I still do not think we are preventing it.

Another item I would like to very briefly comment on is the use of drugs in athletes. Perhaps this is a very common, everyday occurrence, but I think the situations are quite different, when

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you consider the entire picture the commissioners are faced with here. That is, in organized athletics, such as football, there is no legalized pari-mutuel betting, and the public does not have to be protected. They are out there throwing their money up for grabs as they will, but in pari-mutuel betting, the state or whatever unit is controlling it must consider the public as well.

MR. CASSIDY: I would just like to say something that it seems to me I have known for fifty years, and that is that hopping or stimulating a horse has produced sort of hysteria in the minds of the governing bodies, as a rule, when, actually, as far as I know, stimulating a horse does not make him run faster than he can normally, as was said earlier. I think a great deal of hysteria has been the cause of our problems in this.

MODERATOR WOODCOCK: May I just interject a thought in line with what Dr. Oester had to say, and that is this: First of all, he is assuming that every time the chemists are able to detect a specific drug and that drug is found in the saliva or the urine of a race horse, that particular drug should be considered as helping or hindering the movement of that horse. Therefore, his idea is that any time the chemists are able to analyze a sample, and it becomes a positive sample, with the identification of a specific drug, then there should be some rule to cover that. Is that right, Doctor?

DR. OESTER: Yes.

MODERATOR WOODCOCK: What we are trying to bring out here today is that everybody has admitted that there are unethical people available, even in the ranks of the veterinarians, if you will pardon my saying so, except that only once this afternoon was there any questions as to whether the chemist—let's not even use the word "chemist" but let's use the term "analyst" is ever infallible. I think Dr. Oester will agree that it is.

DR. OESTER: I would say it is not infallible. It is subject to error in both directions.

MODERATOR WOODCOCK: It is my opinion that considerable investigation—and now we go back to Mr. Manfuso—is still necessary and is becoming more necessary because of what has transpired here this afternoon.

You realize how necessary it is to determine whether these drugs are doing all the things that Mr. Bruno and Mr. Inglis believe they are doing.

If we could ever get to the point where we could be able to say specifically, and prove to those gentlemen who are the governors of racing, so to speak, that "such a drug will not do all the bad things that you think it will do", then I think we would have more grounds to stand on.

MODERATOR WOODCOCK: We have had considerable discussion this afternoon, and in the Executive Committee meeting we mulled this thing over just the way you gentlemen are doing

it today. Believe me, you gentlemen of the racing commission were well represented at that, too, and so were all other forms of racing, in our argument.

We felt that, since we have had this panel this afternoon and have opened up on you more or less, we thought it might be a good idea, on top of all the criticism, that we offer some pilot instrument, we could call it, by which you could begin to understand the feeling of this organization toward the end that you have in mind. We feel, as practicing veterinarians, that it is much simpler and easier to work under the direction of somebody in the immediate vicinity than it is to work under something that has been established at a distance. Consequently, in the formulation of our pilot instrument, we have put all of the stress and strain and, really, the responsibility squarely on the back of the stewards. So, if you will bear with me, I will read this instrument. If you care for copies, we will see that you get them, and you can work from there. It reads:

"1. No horse to which has been administered any forbidden narcotic, stimulant, depressant, or local anesthetic shall be eligible to start.

"A forbidden narcotic is a narcotic the sale, possession or use of which is prohibited by Federal, state or local laws or regulations.

"A stimulant, a depressant, a local anesthetic shall mean such substances as are commonly used by the medical or veterinary professions to produce such effects, and which are defined as such in accepted scientific publications.

"2. The trainer is responsible for the condition of his horse in any race, regardless of the acts of third parties. He is the absolute insurer of his horse's condition.

"3. No internal medication may be administered to a horse the day of its race without specific written permission from the State or Official Veterinarian after consultation with the Stewards.

"4. Any incident or positive laboratory analysis shall be fully and completely investigated by the Stewards and/or policing agencies of the track. Disciplinary action, at the discretion of the Stewards, shall be taken only **after** investigation establishes violation of this rule."

Now, that is the rule in essence. Added to that: We highly recommend that, regardless of the rule that the National Association of Racing Commissioners eventually evolves, a meeting of the Stewards, Official Veterinarians and practicing veterinarians should take place prior to the inauguration of each race meeting to discuss operational procedures. It is further recommended that subsequent meetings take place as problems arise.

That final paragraph, the recommendation of meetings, is such as happens in the field of baseball where you have national

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rules, but every time two teams meet on a field, the umpire and the manager get together and discuss the ground rules. They might have played over that field a thousand times but they do get together and discuss the ground rules.

We would like very much for you to consider it. We have given you the opportunity to hear from the membership directly, and we think that it may have some value in your next move toward the end that we all want, which is a fair shake for everybody concerned, and a practical rule by which the veterinarian can practice, the trainer can train, and so on.

I think, in the long run, if we can get that kind of a rule, we will have a great many less violations, because the rules that exist today are being violated every minute.

You will have, through the stewards, a greater knowledge of just what you want, information as to the animals that are running, which you can disseminate in any way you want to, as far as that is concerned. At least the stewards at the race track will have knowledge, under this kind of a rule, that they will never have and can never hope to have under a 48-hour prohibitive rule.

MR. BRUNO: Thank you, Doctor. I would appreciate a copy of that resolution. I can promise this convention that the resolution in toto will be submitted for thorough discussion both in the committee and on the floor of the convention. I promise you that.

MR. INGLIS: I also want to thank you very much, Dr. Woodcock, and all the members that are here today. We appreciate very much your work in drafting this proposed rule. I can assure you that, after the Illegal Practices Committee and the Uniform Rules Committee have given it careful analysis it will be given an important place on our agenda at the next national convention. Your organization is cordially invited to be present and to argue in favor of this rule to the fullest extent possible.

MODERATOR WOODCOCK: Thank you very much, Commissioner Inglis.

RECENT EXPERIENCES WITH LEPTOSPIROSIS IN HORSES

DR. DALE K. SORENSEN:

I think, perhaps, a better title to my discussion this morning would be "Recent Experiences with Leptospirosis in Horses." Leptospirosis is a very old disease in horses, and if we consider periodic ophthalmia as a part of the disease syndrome in horses, it dates back many hundred of years. We have to give credit to the Russians for first reporting on this disease, at least reporting a natural outbreak of the disease in horses. I am sure it occurred in this country long before. However, we did not recognize it.

Since the Russians reported this disease, numerous reports have been recorded in the literature on the disease in horses in the United States and many other parts of the world.

Leptospirosis caused by Leptospira pomona in horses has become a well defined clinical entity. Roberts and Bryans—Roberts at Cornell and later, of course, Bryans at Kentucky—have reported on this disease and described the clinical picture rather well.

What I am going to say this morning is nothing new or earth-shattering, or anything like that. We will review some of the manifestations of the disease as it occurs in the generalized form, as well as review some of the forms with periodic ophthalmia, plus reporting on some of the experiences we have had with the disease in outbreaks around the Twin Cities area and Minnesota.

First, a few brief comments on the clinical picture of the generalized disease in horses. It is characterized by very sudden onset. This sudden onset is accompanied by depression. The temperature is elevated, usually 105 or higher. At the same time, the animal goes off feed and gives one the appearance of a really sick animal. At this time icterus may develop. We also might have periodic ophthalmia or iridocyclitis accompanying the disease.

The fever usually persists for four or five days. In the untreated cases it will usually go down to normal. The depression and anorexia accompanies the febrile period. This also will become less severe as the temperature recedes to normal.

As far as some of the other manifestations of the disease in horses, Roberts has reported premature foaling associated with an acute case of leptospirosis. Periodic ophthalmia, also, in the bilateral manifestations, usually occurs in the acute disease.

We had an experience with a case in a pony which developed a clinical picture, very typical of leptospirosis. We had a rising titer on the animal. In addition, the animal started showing signs of colic, or abdominal pain. We operated on the animal

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and found intussusception. The animal died subsequent to surgery, but we feel that perhaps the intussusception was a part of the generalized disease of leptospirosis. Part of this, of course, was just information transposed from dogs. We commonly find intussusception in the dog in the terminal stages. There is no reason why it could not occur in horses as well.

The disease runs a fairly rapid course. At the end of a week the animals usually are feeling much better, with the exception of the periodic ophthalmia. The animals start eating and make a rather uneventful recovery. We will talk a little bit more about periodic ophthalmia later. However, the severe form is an uncommon manifestation of the disease. As you are well aware, serological surveys indicated it is a common disease. Bryans, I think, has made the largest survey or compilation of disease and indicated you can find the disease present in most groups of horses, or 30 per cent of all horses he surveyed.

To give you an idea of what one might expect in some of these outbreaks, I might cite one outbreak we had. This outbreak occurred in a herd of ponies, about 60 ponies. The 28th of November they had a five-month-old pony that was acutely ill. Upon examination, we found an elevated temperature, 1:05.2. The animal was depressed and off feed, very weak. Bilateral periodic ophthalmia was found in both eyes. The animal was also icteric. Examination of the serum bilirubin indicated 2.7 mg. per cent, which is certainly elevated. The hemogram gave a picture of acute leptospirosis, leukocytosis, with neutrophilia, all of the clinical picture that we usually associate with leptospirosis. We went ahead and treated this animal and it made a rather uneventful recovery.

We went back to the original group of ponies and bled them first on November 7. At this time we found 12 of the 60 ponies that had developed a titer against Leptospira pomona, and a week later, November 14, 31 of these animals developed a titer to Leptospira pomona. At this time the owner was getting rather concerned about the outbreak, and she wanted us to go ahead and vaccinate.

We temperatured a number of the ponies and found high temperatures. It had every appearance of going through a subclinical phase, very similar to what we see in cattle. As all of you know, it has been well documented in cattle. Again, most of the cases are subclinical. The subclinical cases may go through a febrile period. However, the animal is not sick enough to be observed. It is a very transient thing, 24 to 48 or 72 hours. These animals are hardly noticed as being ill. We got the same impression in studying this outbreak. No further cases of the acute disease developed in this outbreak. So we just had the one acute case. It is hardly fair to call it a rather severe outbreak. We have serological evidence that at least 31 of the 60 were developing

titers, and I am sure probably most of them eventually did, although vaccination curtailed our activities there somewhat. Subsequent to that outbreak and vaccination, three of these animals developed iridocyclitis. I merely bring this out to indicate that leptospirosis assumes many forms, or many manifestations. The subclinical form is the most common, and it ranges in various degrees of severity up to a real severe outbreak such as Roberts has described in the literature. Most of our outbreaks have been less severe than that, with one or two or three animals involved with the severe manifestations of the disease. Occasionally we will find just one animal in three or four. We have seen more of the cases in the large pony herds or large stables of animals.

As far as the diagnosis of the acute form or generalized form of leptospirosis is concerned, the hemogram is of help. It is different than the hemogram we expect in cattle. We routinely get a leukocytosis with neutrophilia. The white count may get up to 18 or 20 or 25 thousand before it peaks and starts returning to normal. Anemia may also be an accompaniment. However, the anemia is very slight. Many times the dehydration compensates for the anemia, and we have no fall in hemoglobin or hematocrit. This is somewhat surprising since we do get quite a severe degree of icterus in some of the animals. It is certainly a hemolytic anemia, and one would expect more of anemia than you actually see.

Also, serum bilirubin values are beneficial in helping to establish the disease, or to confirm it. But, certainly, the positive diagnosis rests on the isolation of the organism from the blood, or serological work indicating a rising titer. This is not always feasible or possible under all conditions, however. The serum bilirubin values frequently go up to 5 mg. per cent, or at least they are usually elevated.

I might comment on the diagnosis of the acute form. We certainly would like to see an increase in titer. However, this has not always been seen in our cases. In fact, this one acute case that I previously mentioned, this animal never did develop a titer against Leptospira pomona. We feel we have, short of culturing the organisms from the blood, about all the evidence that you could want to make a diagnosis of leptospirosis, and yet this animal never developed a titer.

There are several possible explanations for this. One is, I believe, Roberts' report in his cases that the titer in the treated animals never did get as high as in the untreated ones. It has also been shown in dogs that, by treatment of these cases early, you can presumably kill all the organisms and thus render the animals with no antigen capable of stimulating antibodies, and you do not get a titer. This, we feel, is a good explanation in many of our cases, that the early antibiotic therapy decreases the titer and may actually make it nonexistent.

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Also, we know that the serological tests are not 100 per cent accurate. This has been shown in our laboratory. Occasionally we will take duplicate or triplicate samples and test them on various days and we do get variation, but it is a reasonably accurate test. Certainly, it has to be done carefully.

Those are some of the reasons that, at least, we ascribe to the failure to demonstrate a rising titer or to demonstrate a light titer in some of the real acute cases that have been treated.

Therapy is usually not necessary in the subclinical cases since the disease is self-limiting. In the severe form, it is certainly warranted to treat the animal. We feel that penicillin and streptomycin are probably the most effective antibiotics for it.

Some of the reasons for this is some work we did on cattle, or observations we made in treating the disease in cattle. In cattle we found that penicillin and streptomycin worked very effectively in the subclinical cases that had a high temperature; at least they would bring the temperature down within a few hours.

Giving penicillin and streptomycin will usually cause a rapid fall in temperature within 12 to 14 hours. We also give a supportive therapy. Most cases seem to make a complete recovery.

Now a few words on the periodic ophthalmia and its association with leptospirosis. As you are all familiar, there is a high degree of correlation between the serological titers and periodic ophthalmia or iridocyclitis. This was first shown by some German workers about 1947 or 1948, I believe, and most workers in this country have confirmed this.

As far as our own results are concerned, in general, we find acute cases of periodic ophthalmia that have been going a week or longer invariably show a high titer to Leptospira pomona. We were also able to demonstrate an increase or rise in titer in some of the cases. With the antibiotic therapy we seem to inhibit or decrease the titer. We find that the recurrent attacks invariably have a real high titer at the onset.

In the inactive cases, animals that are blind in one eye, had previous attacks but it is not active at the present time, usually their titers are negative or, certainly, in a very low range, 1:10 or sometimes 1:100.

We do find a few active cases that are not positive and, as I mentioned before, there are several explanations for it. We might also conjecture that other serotypes of Leptospira play a role, or other causes.

Other evidence for the association or cause of periodic ophthalmia from Leptospira, certainly Roberts' reports on the six cases that had acute generalized infection with Leptospira, five of them subsequently developed periodic ophthalmia. One had the periodic ophthalmia during the acute phase of the disease. The other four developed it within a short period of time, but five of the six developed this. Other workers have reported a similar

thing. This also suggests that, in the subclinical form, we could expect the same thing, although probably not in as high a percentage. So, I think we have some circumstantial evidence here.

We also have reports of the isolation of a strain of Leptospira from a clinical case of periodic ophthalmia. However, this claim was not well founded and not generally accepted to be extremely valid.

We have other indirect evidence that we might look at. One is that humans with leptospirosis will frequently develop iritis or iridocyclitis in the acute phase of the disease and, also, will develop iridocyclitis subsequent to recovery from leptospirosis. We also know that on one or two occasions iridocyclitis has been observed in cattle when they were experimentally infected with Leptospirosis Pomona.

In some work with dogs at Minnesota, Drs. Low and Mather found, that three of ten dogs developed iridocyclitis with experimental infection of Leptospira icterohaemorrhagiae.

On the other side of the ledger we have the theory that periodic ophthalmia hypersensitivity, which is a very good theory, that the uveal tract or iris or ciliary body has been sensitized to Leptospira and in subsequent attacks, as a result of re-infection, which more or less challenges these tissues, you get the reaction. This is certainly not unfounded. There is some evidence on this side, also. One is, the failure to isolate the organism from the acute cases suggests that this might be true. Also, we know that any sequela to a disease may be associated with a hypersensitivity. We think of nephritis as a result of streptococcal infections, or rheumatic fever, and so on.

We also know that many workers have succeeded in producing iridocyclitis by sensitization with various antigens, such as protein, and so on. This could be done with rabbits and other animals.

It has been demonstrated that agglutinins are present in the affected eye, whereas not in the unaffected eye. This may lend support. On the other hand, we know in an animal with iritis, the permeability of the vascular system in the iris is not particularly good, and protein and other things leak out. So, finding agglutinins there does not necessarily mean, of course, it is a result of hypersensitivity. I think, in the final analysis, there is some evidence both ways, and we cannot say, at the present time, whether is is leptospiral infection per se or whether it is a result of hypersensitivity.

I might also point out that leptospirema is difficult to ascertain in the acute cases. The organisms are present in the blood for a rather short period of time and may be just getting in too late into the eye to recover the organism. So much for some thoughts on the cause of periodic ophthalmia in relation to leptospirosis.

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Now a brief review of the clinical picture of periodic ophthalmia. Certainly it is an iridocyclitis. The animals usually do not show any particular general signs or general manifestations of the disease. They are somewhat depressed. However, the TPR is normal, and perhaps the depression is associated with the pain and the photophobia one sees in the eye. The disease is ushered in usually by photophobia, a little excess of lacrimation of the affected eye. Upon examination of the animal, one will usually find a decreased intraocular tension. The animal is very sensitive to light, and it keeps the affected eye closed most of the time. The pupil is constricted down, and iritis is usually evident, particularly in the acute phase. The iritis is characterized by the iris being thrown up in the folds, giving sort of a velvety appearance. At the time we might see a ciliary flush and injection of the vessels around the limbus. Several days later in the disease we may also start seeing hypopyon, presence of pus and protein into the anterior chamber, which usually settles down to the bottom. This may be followed by cataract or opacity of the lens, because the lens gets nutrition from a normal aqueous. The lens starts becoming opaque. When you look at it with a pen-light or ophthalmoscope, you get a greenish cast or haze to it. You cannot see through it, at least not clearly. You cannot see the posterior segment of the eye. Also keratitis may start developing at this time.

In the untreated cases, several things can happen. One is that adhesions can form between the iris and the lens, known as posterior synechia. You will also have adhesions of the iris to the cornea, known as anterior synechia. Of course, cataracts might develop, and you might have residual opacity of the cornea. You might have a sunken and generally fibrotic eye. If we can have the slides we will try to briefly show some of these lesions.

[Slide] In this picture we are attempting to demonstrate the decreased intraocular tension. Certainly, it is very important to stand directly in front of the horse to visualize this. Notice the size of the palpebral fissure on your right side here. The other side is the affected eye, with the decreased intraocular tension. Of course, the whole eye is sunken in and the lid collapsed. It is a very good, early sign of periodic ophthalmia.

In regard to the therapy of periodic ophthalmia, again, this is well documented. A standard procedure has been followed by the ophthalmologist for many years, and much of this has been adapted to veterinary medicine in the treatment of periodic ophthalmia.

Certainly, it is desirable to place the animal in a darkened stall to lessen the photophobia and afford some relief to the animal. Secondly, it is equally desirable and imperative, particularly in the acute cases, to dilate the pupil with atropine. We usually use 1 per cent solution of atropine, giving 1 to 3 drops, or at

least one drop three times a day to keep the pupil dilated. As most of you know, this is to prevent the adhesion of the iris to the lens or to the cornea. Also, hot compresses are desirable to relieve some of the pain, and it keeps the owner busy, and he realizes he has something to do with the case.

In addition, we have been putting these animals through a rather intensive course, of penicillin and streptomycin for at least four to five days, sometimes six or seven days. We are doing this for two reasons. One is in an attempt to sterilize the animal, so to speak, and prevent the recurrent attacks. This is based on the assumption that periodic ophthalmia is leptospirosis per se and not a hypersensitivity. We feel that we get good responses in the acute cases. If these cases are obtained soon enough and treated, recovery is the usual rule, complete recovery, with no residual damage, such as adhesions or cataract or keratitis.

In addition, many times we use local treatment on the eye, in the form of cortisone applications or chloromycetin. Chloromycetin has been shown to be the most penetrating of all the antibiotics. It is the antibiotic of choice to use locally in the eyes. Cortisone is used to prevent pannus. It is usually helpful. However, it is not particularly necessary in many cases.

We have been trying to follow our cases of periodic ophthalmia. We followed some of them as long as four years. So, it ranges all the way from a few months to four years. We have not had any recurrent attacks of iridocyclitis in these horses.

These horses consist of animals treated in the acute phase, some already blind in one eye but had recurrent attack in the good eye, and a few of them were animals that did not have the active disease. They were already blind in one eye, but the owners wanted to try to prevent recurrent attacks, so they wanted them treated.

To date we have not had any recurrent attack in any of these animals, so we are somewhat encouraged. We, of course, realize this is a very sketchy thing and, certainly, one cannot form any valid conclusions yet for quite a number of years, and more cases are necessary.

This is somewhat contrary to Roberts' report where these animals were all treated and yet did come down with periodic ophthalmia subsequent to the acute leptospirosis. Nevertheless, we feel that we are getting some results.

We had one case that came back with a recurrent attack. We went back to the records and found that this animal was not treated the first time, so I felt a lot better about that one.

The titer in these animals seem to go down essentially to nothing, or become negative within a few months after treatment, and most of them have remained so.

We have had one animal, however, where the titer became negative and about four or five months later it rose again and

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went up to 1:10000 and higher, and stayed at this level for a couple of years. When we treated it with antibiotics, the titer went down again. This animal has not, however, had any recurrent attacks. On the other hand, we mostly find these animals do have a low titer or no titer at all due to Leptospira pomona, subsequent to treatment, or subsequent to their acute attacks.

There certainly are many unanswered questions on periodic ophthalmia and leptospirosis in horses. I think we have touched on a few, but we have many, of course, left to work on. Why does it only occur in one eye, and conclusive proof that it is leptospirosis per se, or hypersensitivity, and many other questions.

Thank you very kindly for your attention. [Applause]

CHAIRMAN WOODCOCK: Thank you very much, Dr. Sorensen.

Are there any questions you might care to ask Dr. Sorensen right at the moment?

DR. E. E. EBERT [Columbia, Mo.]: Do you have any information on how long the untreated animals go through the disease and remain carriers?

DR. SORENSEN: No. As far as the carrier state of leptospirosis, I do not believe anybody has demonstrated that horses do, in fact, become carriers. There has been some work done on the urine shedders and localization of various tissues. It is pure conjecture that they do become carriers. No one has ever demonstrated that.

DR. C. H. BURGER [Bakersfield, Calif.]: Do you use a plate or tube test, and what is the significant titer?

DR. SORENSEN: We use both the agglutination lysis test and plate test. The agglutination lysis test is more sensitive in our hands than the plate test. For example, in this one outbreak I cited, the first one, only one horse was positive on the plate test, where twelve were positive on the agglutination lysis test. In general, we feel we get a titer of 1:100 or 1:1000 on the agglutination lysis test before the plate test becomes positive.

As far as what is the significant titer, certainly it has to be correlated with the cause of the disease at that time. If you are dealing with an outbreak, of course, it is rather easy to evaluate. If you are dealing with an individual animal, it is, again, very difficult to evaluate.

As I pointed out, in the chronic cases, we do not find any that are inactive. If they are active in the recurrent attacks, we find a high titer, which is significant. In the acute cases, if we let them go untreated, they usually demonstrate an increase in titer. Certainly, any increase of tenfold dilution, 1:100 or 1:1000 or 1:10000 is significant. On the basis of one test, it is difficult to evaluate.

DR. JACQUES JENNY [Unionville, Pa.]: I have loaded questions. First, in your lysis test, do you check just for pomona

or other Leptospira as well? Secondly, how would you explain that the horse that had several attacks, the horse that was treated in more or less the manner you outlined, possibly with more steroids at the time of the acute attack, to forestall adhesions and what-not, every time that horse was subjected to an extreme stress he came down with another attack. That would not fit into an exposure to infection. It would rather tend to your second possibility of sensitivity.

DR. SORENSEN: On the first question, we checked for icterohaemorrhagiae along with the pomona. We usually find a low titer in all of these icterohaemorrhagiae.

I do not know how you can explain the recurrent attacks. Perhaps in some cases it is a hypersensitivity. I might cite one example in a herd we vaccinated. Following vaccination, we had three or four outbreaks of what looked like iridocyclitis, but they were not the full-blown or real acute form. This occurred within three or four weeks after vaccination, when they should have had a real high titer, yet we had the three or four cases. We were wondering if we did not sensitize the animals, or challenge with the inoculation.

I certainly cannot answer why stress should precipitate these attacks.

DR. C. E. EASTIN [Lexington, Ky.]: Would you comment on your experience with the vaccine and what value you think it has, and possibly any untoward reactions? You may have already touched on that.

DR. SORENSEN: We have not used the vaccine in too many outbreaks. As far as its merits are concerned, I think it has been well demonstrated it does afford some protection, probably lasts for six months, or longer than that. I feel that in most cases, by the time that you get in there, except in real severe outbreaks, the disease will have already spread through most of the animals and it probably will not be necessary.

Leptospirosis in horses is largely a subclinical disease, and it is only occasionally that we see acute generalized form, or only occasionally that we see periodic ophthalmia. I think all surveys have indicated it is an extremely common disease of horses, and it is most apt to occur in army remount stations or any place where horses are assembled from various, different places. It seems to be a rather mildly contagious disease but will sweep through large groups of horses.

We see more of our cases in these large farms that have many horses, that are buying and selling horses rather frequently.

DR. RICHARD GILYARD [Waterbury, Conn.]: Although it is regarded that riboflavin studies might have been a little premature and, perhaps, the admixture of riboflavin nowadays has been discarded as a means of preventing periodic ophthalmia, it may be coming back into the picture, from a deficiency angle,

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which will permit and does permit the Leptospira to take on clinical severity in horses in the form of periodic ophthalmia.

In Connecticut, we find a great many horses carry a titer to Leptospira antigens, likewise a great many to Brucella antigen.

In response to Dr. Jenny's citing cases which were recurrent in antibiotic therapy, we had such a case, and that horse simultaneously carried a titer to Brucella.

The farm outbreaks of periodic ophthalmia in New England, under adverse winter feeding and adverse winter climate conditions for young horses, suggest that perhaps we have to tie back into a weakening influence which may be related to the riboflavin theory.

DR. SORENSEN: I have no particular comment on the riboflavin therapy. I would point out in cataracts, the lens itself has a high content of riboflavin plus vitamin C and other proteins. In the eye with a cataract in it, the concentration of riboflavin, vitamin C and these other preparations is considerably reduced, suggesting that possibly there is some association with riboflavin deficiency.

A PRACTITIONER'S EXPERIENCE WITH INFECTIOUS ANEMIA IN TEXAS

D. J. Anderson, D.V.M.

Thank you, Dr. Woodcock and gentlemen. Although we are running a little late, I am placed somewhat in the position of the speaker who rose and said, "Before I start my speech, I would like to say something." [Laughter]

Now to the subject at hand, a discussion of equine anemia, clinical cases which we were fortunate or unfortunate enough to see in our practice this past summer.

In considering this problem, a few questions have arisen in my mind, not to be answered here necessarily, but for consideration. First, is infectious anemia a big problem? Second, how extensive is it? Third, does the average practitioner use proper precautions? Fourth, how might information regarding infectious anemia affect our insurance picture? Fifth, what is the attitude of the average owner when he has an infectious anemia problem? Sixth, why have facilities at the University of Miami not been utilized to a greater extent in the study of this disease?

Seventh, do owners want to know all about their horses when trouble arises? Eighth, do we feel that the test perfected at the University of Miami is reliable?

As stated, these are some of the questions that have entered my mind regarding this disease. The problem which confronted me was in two thoroughbred brood mares.

The first case occurred about August 1, 1959. The blood showed a rapid sedimentation rate. There was a history in this first case that a blood transfusion had been given this animal about thirty days prior to our entry into the problem. This animal showed weakness in the hindquarters, and ecchymotic hemorrhages along with anemia. Drawing a blood sample from this animal it had the watery look that you have heard of. It continued to eat fairly well. Temperature was about 103. Animal showed severe emaciation. These, of course, are typical clinical symptoms of this disease.

We were a little suspicious in the beginning but placed the animal on supportive treatment, antibiotics, aricyl, and a little vitamin B therapy.

Five days later I was called. The temperature was still 103°. So that made me more suspicious than ever.

Another mare was brought out of the pasture August 20 looking like a so-called gutted snowbird. Of course, both of these cases really looked ready for the glue factory. We contacted Dr. Teigland. Blood samples were forwarded to Dr. Saurino at the

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University of Miami. Dr. Saurino ran tests and reported these samples showed the strongest positive reactions he had seen. He was happy that he had found some field cases to work on. These were the first field cases that had come to his attention.

Although the money for the project down there had been expended, it was his desire to run blood samples on the rest of the mares on this farm. I assured him of our full cooperation and thought that we would be sending him some more blood samples right away.

On contacting the owners, they were rather uninterested, a little vague. They remembered the New England trouble. They realized the seriousness of the disease but they just never did get around to having us send blood samples on the rest of those animals for laboratory examination. People just seem to refuse to face this problem, for some reason.

In talking with Dr. Teigland, he ran into the same problem in Florida. This can be, as I see it, pretty serious, because, quoting one of General Kester's statements not long ago, the average horseman today has "shot" phobia. If he has a little pathological condition, a little trouble with the horse, he thinks the doctor can give him a shot, and that is all that is necessary. He does not see the necessity of making these thorough examinations. But this shot phobia thing can be dangerous, because improper use of hypodermic needles, and the like, by the laity or those not so conscious of the problem, can certainly get us into more trouble than we are already in.

Now what I am wondering, is everyone seeing equine infectious anemia and kind of forgetting it or, as Dr. Teigland says, sweeping it under the rug? I don't know but, if this is the case, I think this is the place to give the problem a little closer look.

Many veterinarians who do not do too much equine practice might not be fully cognizant of the dangers of transmission spread.

Another case which I would discuss briefly, and in which I possibly rather strongly suspect infectious anemia was a thoroughbred gelding that originated in California, came to the tracks in Arizona with a history that they would get him near ready to go and he would go lame in one leg. Take him out a while and bring him back, and they just never could get him ready. By the time he got home, he was in very poor condition. That was about last June. We took blood and did a little blood chemistry. Everything looked all right, except he was very low in vitamin A and was rather anemic. Ran about 4 million red blood cells. We were very happy that we had discovered what was wrong here.

We could see that this horse had been kept in a dark stall at the track and had been fed prairie hay and shelled oats, which was an ideal setup for vitamin A deficiency. We placed this animal on vitamin supplements and it came along very nicely, but we still haven't, even with other treatment, been able to get the red

blood count above 5 million on that horse. Is this one of those so-called recovered cases of infectious anemia? How many of us are running into that thing and possibly overlooking it? I don't want to be an alarmist. We want to be practical in our approach to these problems, but I am reminded—pardon me for mentioning the great state of California and their problems, because we do have a few problems in Texas, too—of the vesicular exanthema in swine which existed in the state of California for twenty years, and we might have had it in our other states, too but we kind of swept it under the rug. When it flared up, we really had trouble.

So, gentlemen, if we have some infectious anemia in Florida and have some infectious anemia in the great state of Texas where the sun shines and it is such a healthful condition [laughter] it is reasonable to assume that we may have it other places.

Back to this matter of laboratory work: a positive report is always welcome, or even a negative report. We feel, in the field, if we can get a little laboratory help on some of these clinical problems, it helps us a great deal.

In conclusion, I would like to mention about three things. As I see it, the test for equine infectious anemia developed by the workers at the University of Miami has merit. They do not know how reliable it is until more work can be done. Someone should find the way to finance that work and have it continue.

Thank you. [Applause]

CHAIRMAN WOODCOCK: Thank you very much, Dr. Anderson.

Were there any questions that anybody would like to ask Dr. Anderson?

DR. A. W. PATTERSON, JR. [Hanover, Pa.)]: I would like to ask, in the clinical cases, how many mares were there where he picked up the original two? How many more showed the clinical symptoms?

DR. ANDERSON: We only know of the two that showed clinical symptoms. There were about 15 or 20 more mares on the farm.

DR. PATTERSON: That would necessarily indicate it is not a highly infectious disease.

DR. ANDERSON: That I do not know, because we do not know how many of those animals may be carriers, may have apparently recovered from the infection. That is, to me, a question that requires more study, more work.

DR. D. K. SORENSEN [St. Paul, Minn.]: I can mention that during the past four or five years we have had four cases of infectious anemia in the state of Minnesota that have been confirmed by animal inoculation into the horse. So, we are seeing it up there, and I am sure we are missing a lot of cases, too. We have had four that have been confirmed.

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DR. ANDERSON: We are sorry we failed to make animal inoculations on these cases. We had the facilities and all, but in a busy practice we just stump our toes on those things, sometimes.

CHAIRMAN WOODCOCK: As a follow-up to Dr. Anderson, Dr. Teigland will describe some of the most recent work on this particular condition.

REPORT ON RECENT WORK IN INFECTIOUS ANEMIA IN THE HORSE

M. B. Teigland, D.V.M.

I would like to say, that the funds for any research work have been expired for three years, and if it were not for Dr. Saurino and the other men at the University of Miami and the attitude of the dean, there should be no furtherance of this research program at all. There are no funds. Dr. Saurino has received no reimbursement for any of his work since this project was begun. He does not seem to request this reimbursement, but I do not think this is right.

This summer we again went through some infectious anemia in Florida. I would like to emphasize one point. I have never seen a case of infectious anemia on a race track in Florida. We all know about the New England outbreak. We all know the history of how this took place. It is for this reason, if there is any infectious anemia left in the United States, this again is possible. The hypodermic needle only requires that it be injected into one horse carrying the virus which he apparently carries for life, with or without symptoms. This needle, without heat sterilization, will reintroduce the infection by merely introducing it into another horse, which we assume are all susceptible. Therefore, it has its implications.

We have infectious anemia in Florida pretty well statewide. There have been so many cases this summer, over the state, that Dr. Saurino has been flooded with phone calls and has been embarrassed because he could not come up with even the time to do the test. But in some way or other, working at night and whatnot, he ran the tests I am going to describe.

I am going to read the paper on the recent work that has been done by Dr. Saurino:

"Two years ago this group reported the progress made toward the establishment of a laboratory diagnostic method specific for equine infectious anemia. We indicated at that time that our approaches to this problem from the fields of embryology, tissue cultures, hematology and pathology yield reactions to positive horse serum, but the tests were either nonspecific or cumbersome and technically fastidious.

"During the past two years, our efforts have been concentrated on two approaches: one in immunology and the second in enzyme chemistry.

"The precipitin reaction: It was felt that a source of equine infectious anemia virus was essential for the development of a specific antibody. Toward this end, it was assumed that the most

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readily available source of this virus would be the blood system of the infected horse.

"Rabbits were injected with washed red blood cells of clinically positive horses, and an equal number of rabbits were immunized to the positive horse plasma. Control rabbits were used by inoculating with cells and plasma from a clinically negative horse. Four immune serums were attained, in this fashion, from the rabbits:

"1. Anti-positive cell.
2. Anti-positive plasma.
3. Anti-negative cell.
4. Anti-negative plasma.

"Each of these antisera responded to a high titer when used against its specific antigen. There was considerable cross-agglutination in the case of the anti-positive cell serum and negative horse cells. The same was true in a precipitin reaction when crossing the anti-plasma rabbit serums and their antigens. The titer of the antiserum to its homologous antigen, however, was always greater. In other words, the anti-positive horse plasma rabbit serum gave a very high titer against positive horse plasma and a lower titer against normal horse plasma.

"It was most interesting eventually to find that the anti-positive cell rabbit serum precipitated, to a high titer, the positive horse serum and gave practically no precipitate with negative horse serum. Here we see a peculiar phenomenon where a rabbit's serum immunized to positive horse red cells now reacts in a precipitation with positive horse serum. Our only explanation of this would be a guess that the virus of infectious anemia or some red cell entity is liberated into the serum while an animal suffers from the disease.

"We have used this precipitin test, anti-positive horse red cell rabbit serum, to test some fifty serum specimens arriving in our laboratory from field cases from various parts of Florida. In every instance thus far, our results indicated reliable reactions to positive sera and no reactions to negative sera. Many cases, when tissues were later submitted, were verified by pathological tissue findings.

"We cannot say at this point that we have developed a specific test for equine infectious anemia. We feel sure, however, that there are merits to this approach and that more cases should be analyzed and more diseased organs used for the extraction of virus.

"Studies with aldolase: The enzyme, aldolase, is present in all tissues, and only when those tissues are disrupted does one find a large quantity of this enzyme free in the blood stream.

"In our cases of equine infectious anemia we found a steady rise in aldolase as the disease progressed. We find that normal values for horse serum run from 8 to 20 units. In swamp fever,

these values rise. We have seen some samples yield 50 to 60 units of aldolase.

"The test itself merely measures the suspect serum's ability to digest the substrate, fructose-diphosphate. This is then converted into aldolase unit values."

DR. TEIGLAND, continuing: It is a very difficult thing to expect a man in the laboratory to say that he has a good test, working from unknowns. We have to have knowns, and this has to be done on a research basis. The facilities have been available. We have had a place where we can reproduce it. It has been approved by the state. We have room for two horses that we can at any time infect. This costs money, as you all know. We are faced with the same problem as Dan. When you get a case of infectious anemia, are you going to be able to animal-test this case to prove in every case that it was infectious anemia?

The big concern I have is not the horse on the track, except we have ponies on the race track, and where do they come from? This pony might be purchased in an area of Florida where we know we have it. If he is, how much regard do we give the pony on the race track? If he is not doing too well, oftentimes the trainer does not say too much about it. The only fortunate thing is probably he is not needling the pony. [Laughter]

Regarding insects, we have pretty good control on the race track, and we must keep it that way.

Gentlemen, that is about all I have to say. If anybody wants to see this movie, if there is any place or any time it can be arranged, I will be happy to show it.

Thank you. [Applause]

CHAIRMAN WOODCOCK: Thank you very much, Dr. Teigland.

Is there anybody in the audience who would like to ask Dr. Teigland any questions on this particular phase?

DR. J. C. HANSEN [Beaumont, Calif.]: I would like to make a comment and ask a question, too. In 1954 or 1955, I do not remember which, I came into contact with a quarter-horse stallion who had been at the race track. The fact of the matter is he was just between race meets and was getting a little home care and freshening up before he was to return to Bay Meadows again to race.

New Year's Eve, as I was getting ready to go to a New Year's dance, the phone rang. I told my wife I would see her later. I went out and examined the horse and, to be honest, I was not sure what was wrong with the horse, but it seemed like one of these cases of atypical type of pneumonia or pleurisy or some such.

The case progressed over a period of three or four days. Reviewing the history I became very suspicious of infectious anemia.

In the meantime, the animal apparently made a recovery. I advised the owner of this horse that I still was not positive he

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did not have the so-called slime fever. I said, "If this horse should recur with the fever in a period of thirty days or so, be sure and get the horse to the University at Davis and let them do some animal experiments."

In about three weeks, the animal ended up at Davis and was subsequently destroyed. Dr. Wheat can elaborate more on what they found in the horses inoculated and, also, at autopsy.

This horse had been at the race track, Bay Meadows, San Mateo, California. It caused quite a little stir momentarily, in that the veterinarian who represents the Los Angeles Turf Clubs at Santa Anita and Hollywood Park made a trip to Davis to find out where this horse had been and what possible contact could possibly have been made.

So, there may be a problem due to transportation, and such, on a race track.

Since that time I have had occasion to suspect infectious anemia in harness horses who came from the East Coast where there could have been contact. Two separate occasions, two consecutive years, these horses showed symptoms which I was not able to positively diagnose. They showed icterus, showed a low red count, with a lack of elevated white count. Temperatures were low, 102. They did not have what you would call a real high fever, but there was a little fever.

I became suspicious of these animals, and the fact they were at the race track where a race meet was in session, I was very reluctant to make very much noise about it. The trainer and owner of the horse, the management of the race track and myself got together, and we isolated the horses on the track, screened off the stalls for insect control. Between the three of us, we got a horse which I personally knew was free of the infection, because I had known the horse, knew his surroundings. We did animal inoculation at the race track, kept the horse in confinement for a period of approximately 50 days. There was no subsequent following disease.

What I am wondering is if this is a good way or a bad way to handle suspicious cases where you are not sure, where you have a subclinical case, if it were proven, or if you should make enough noise about it to bring in the authorities and possibly quarantine the race track. Or just how would you recommend that it be handled?

DR. TEIGLAND: In the first place, this is not a contagious disease; it is an infectious disease. I think, if we as a group can control the use of hypodermic needles, there is no problem on the race track with infectious anemia. I would not be afraid to have a horse with infectious anemia in a stall next to another horse. This has been pretty well proven. The urine carries the virus and fecal material carries the virus. There have been occasional reports in the literature that this has been transferred orally. It

probably involved spots or open lesions for it to enter the blood stream. You apparently have to contact the blood stream to reproduce this disease.

I think the hysteria that arises, as it did in New England, was due to improper use of hypodermic needles. I do not think we have too much worry if we can control this. We still need a test for the cases when they come up. We still need a way of eliminating the carriers that are around the areas where they exist.

DR. J. M. BREWER [West Chester, Pa.]: Are you minimizing the possibility of infection by insects, or passage?

DR. TEIGLAND: If so, I'm sorry. I didn't mean to. In the areas where we are seeing it, out on the farm, out in the swamp areas of the outlying lakes, this is the entire problem. Any severe outbreaks I have seen have positively been insect-borne.

On the race track, in most cases, at least in Florida, the insect control is very good. In the wintertime we have hardly any of the true biting insects.

DR. H. M. SMITH [Hanover, N. J.]: Is it not possible, in view of the shortage of funds, and the exhaustion of funds by Dr. Saurino, could a laboratory fee be established for this work, whereby any time we send a sample down there for testing, we will more or less be able to hold our own end up, not being harsh on you or the laboratory?

DR. TEIGLAND: Let me tell you the problem involved in this. This has been thoroughly discussed. You cannot run a test such as this without full-time help ready to do the test. Dr. Saurino is a very busy man. If we had enough money to work full-time technicians who would be available twelve months of the year and could be assured of their job, they could be taught the technic and be available to do the work. That is the only way it can be done. We need hands. We also need a proven test. We think there are many more approaches that will work well.

DR. J. A. SCHAUB [Eau Claire, Mich.]: How does this test compare with the Japanese test?

DR. TEIGLAND: Talking about the hemosiderin test?

DR. SCHAUB: No, the Japanese test that has few inclusion bodies in the periphery of the cells.

DR. TEIGLAND: I think the last I read on the Japanese work is that the standard test in Japan at this time is the biopsy of the liver and examination for hemosiderin.

This blood has been examined by one of the hematologists in connection with the Medical School down there. He has seen nothing about these blood cells that would indicate this is true. We have nothing to verify that.

DR. SCHAUB: This test is Upjohn's. They just make a smear and pick out the blood cells that are affected.

DR. TEIGLAND: We have not made this approach at all.

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DR. SCHAUB: They even had a Japanese fellow at the college running some of them.

DR. TEIGLAND: Did they think it was effective?

DR. SCHAUB: Yes, they did. I wondered how it compared to yours.

DR. TEIGLAND: I don't know. I would be interested in having some knowledge of this test.

DR. SCHAUB: Dr. Stoner and I have had thirteen or so cases this summer.

DR. TEIGLAND: In Michigan?

DR. SCHAUB: I am probably the only veterinarian here that owns a horse with it. [Laughter] I leased a stallion from a fellow. He had six mares, and all of his mares have contracted the disease. My stallion has and all the mares he has bred this summer. We have traced them down, and most of those have had it.

DR. TEIGLAND: What did you do about it when they have had it?

DR. SCHAUB: Most of these horses, outside of the ones that have died, have recovered.

DR. TEIGLAND: Are they carriers?

DR. SCHAUB: Who knows?

DR. TEIGLAND: Have they been tested?

DR. SCHAUB: We have sent the blood in on the recoveries, and they are all right.

DR. TEIGLAND: All right by test or animal inoculation?

DR. SCHAUB: We haven't animal inoculated. According to this blood test, the blood has cleared up.

DR. TEIGLAND: I would hate to own the horse you give the blood to, because I have not yet found a horse that had infectious anemia that was not a carrier. If you remember Mott working at Beltsville, Maryland. He worked with this thing for twenty-five years before he retired. The last time I talked to him he had two horses at Beltsville that had been there for nineteen years, and perfectly normal, but were isolated and screened. Any time he wanted a case of infectious anemia, he took a little blood and reproduced it.

COLICS; THEIR CAUSE AND TREATMENT

A PANEL DISCUSSION

Moderator: W. F. Riley, Jr., D.V.M.

G. H. Keown, D.V.M.

J. D. Wheat, D.V.M.

O. R. Adams, D.V.M.

MODERATOR RILEY: We have decided this morning to split our talks up, based upon the films that Dr. Adams and Dr. Wheat have to show.

I will briefly describe the general principles of medicinal treatment in the handling of colic cases. A review of the records of our ambulatory and hospital clinics show the incidence of equine indigestion over the past several years to be in the following order as to occurrence:

a) Flatulent colic
b) Spasmodic colic
c) Impaction colics
d) Engorgement colics

The highest mortality rate has been among the latter two types due to rupture of the affected organ. Death was infrequent in intestinal and impaction colics where professional aid had been sought early and prompt treatment instituted.

The diagnosis must be based upon an accurate history with particular reference to feed and recent physical activity, plus a careful physical examination. A rectal exploration should always be made. The latter gives the examiner a knowledge of the amount and consistency of the bowel contents, and the degree sensitivity, tension and gas distention. Any obvious impactions or displacements can be detected.

Principles of Treatment

The principles involved are the control of pain, evacuate the digestive tract (in most cases), and to re-establish normal peristalsis. To accomplish this, one has a variety of agents to choose from. Although several may demonstrate a desired end result, the manner in which the action is accomplished may be deleterious on a particular case. It is important to choose the drug which will achieve the proper action with the least embarrassment to the patient.

Control of Pain

In less violent flatulent and spasmodic colics and in impactions, I prefer to administer chloral hydrate by means of the stomach tube at the same time other medicinal agents are ad-

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ministered. Usually within 15 minutes the effect is noticeable. There is available commercially a chloral hydrate—potassium bromide* compound that has been very useful for many years. In more recent years, Demerol hydrochloride** and Novin*** have proven useful on the more violent types in order to control the patient and allow passage of the stomach tube. Demerol has the disadvantage of requiring a narcotic permit, but enjoys a slight edge over Novin (Methampyrone) in its spasmolytic and analgesic action. In the more violent types of colic it is the first agent administered to gain control of the patient for the balance of the medication.

For many years, atropine sulfate (¼-½ grain) was used to relieve intestinal spasm and still is useful. It, however, stimulates the respiratory centers, and accelerates heart action (by depression of the terminations of the vagus), which often is undesirable. In less severe flatulent and spasmodic colics, it is still employed successfully by some practitioners.

The tranquilizers have not been used to a large extent in our clinics for colic. In a few instances where they were employed, the results were variable.

Evacuate the Digestive Tract.

I have never quite felt that I could always accurately judge which serious case of colic would stand a rapid-acting peristaltic stimulant such as arecoline or in more recent years Carbachol (Lentin). They are definitely contraindicated in engorgement colic, finding their greatest value in impaction and mild flatulent colics. The administration of adequate amounts of mineral oil as a basic laxative agent or as second choice a hypotonic solution of one of the hydrogogue cathartics, is still our favorite approach. After the oils have had sufficient time to lubricate and soften, Lentin usually works very well. It may be employed using half doses on many occasions. More recently, the emodin cathartic cascara sagrada in the injectable form (Parenterin and Peristaltin) makes a safe and effective augmentation to the liquid paraffin. There is less intestinal spasm and the horse tolerates the action well. Superpurgation has not been a problem.

Twenty years ago, aloes and Aloin were commonly used to evacuate the digestive tract. Due to their often unpredictable time of action and possible side effects, they are used less today. Raw linseed oil is also apparently less popular now than in former years.

In an attempt to re-establish normal peristalsis, various carminative and antiferment preparations are routinely used. We

*Chloral compound with potassium bromide, Pitman-Moore Co.
**Demerol hydrochloride, Winthrop Laboratories
***Novin, Haver Lockhart

frequently use one containing a mixture of Camphor, Salicylic acid, Oil of Turpentine, Capsicum and Ginger (Turcapsol, Pitman-Moore). It is convenient to use such a compound and it is effective. It is useful in all forms of colic, but in flatulent cases they act as a stimulant and carminative and as such seemed to perform adequately in the average colic case. I still use them occasionally on milder types of colics, mixing them with mineral oil and administering by the stomach tube. Coecolysin Bengen*, an organic extract from parts of the intestinal tract has come into being in the past years as a peristaltic stimulant. (Limited experince with this agent has indicated it may be useful to stimulate intestinal secretion and evacuation in all forms of colic.)

Severe indigestions, particularly from succulent or spoiled feeds warrant detoxification procedures. The intravenous injection of dextrose, as well as Infusion Hydrocortisone and Antihistamines should be administered to prevent possible complications from laminitis.

Repeated attacks of colic over a period of weeks or months are regarded as unfavorable. Commonly they may be the result of one of several causes as follows:

1. Persistent impactions, particularly the cecum.
2. Enteroliths, or phytoliths causing colon blockage.
3. Parasitic Aneurism of mesenteric artery.
4. Chronic gastric dilatation.

Aftercare following severe colic attacks should include rest, limited food intake, free access to water and the correction of any obvious irregularities of feeding or management that might occur.

DR. G. H. KEOWN: My section is on spasmodic colic. This form of colic is a very common one. Kral and LaGrange in their book on "Colics of the Horse" state that it constitutes probably 30% to 40% of all colics. It is due to a spasm of the intestines, that is, a very strong contraction of the musculature of the intestines.

Kral and LaGrange discuss a number of theories that have been given to explain the causes of spasmodic colic. One, is that it is due to a catarrh of the intestinal tract. Another, that it is due to stimulation of the vagus nerve, creating a condition that is spoken of as vagotonia.

Still another theory is that it is due to atmospheric changes. If there is a sudden change in the weather or a sudden change in the barometric pressure it has been found that there are more cases of spasmodic colic then at times when the temperature and barometric readings are steady.

Proponents of the theory that temperature is important in the production of spasmodic colic point out the fact that colic will

*Coecolysin produced by Bengen & Co., Hanover, Germany

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often develop following ingestion of cold water particularly when the animal is hot. Also the ingestion of frozen foods, such as, vegetables, can bring it on.

Other causes have been reported by various authors, including the ingestion of moldy straw or grain et cetera. Probably all these different causes are due to the stimulation of the vagus. So, vagotonia takes place in all cases regardless of the actual exciting cause that may bring it on.

The motility of the gastrointestinal tract is controlled by the autonomic nervous system. This system, the autonomic nervous system, innervates all involuntary muscles and, also, the glands of the body. It consists of the parasympathetic and the sympathetic nervous systems which are antagonistic in effect. The parasympathetic nervous system supplies stimulatory activity to the gut, stimulating the muscle, while the sympathetic has the opposite effect. It causes inhibition of the muscle and relaxation of the intestinal tract. Neither of these carry any sensation of pain. The sensation of pain is mediated by the visceral afferent nerves, and they set up the chain reaction.

Nerves in the autonomic nervous system act through ganglia, so that both preganglionic nerve cells and postganglionic nerve cells are found. This is a different arrangement than is found in the voluntary nerves where a direct nerve connection from the central nervous system to the muscle end-plate exists.

Horses are very sensitive to pain, more sensitive than other animals, and the action of pain causes marked behavior reactions that we recognize as colic.

The stimulation of the visceral afferent nerves causes a reflex stimulation of the vagus, which then, in turn, brings about a contraction or peristaltic wave. The impulse may be initiated by fermenting food with the production of irritating materials such as fatty acids, carbonic acid ormethane, et cetera. If the resulting peristaltic-like contraction is vigorous the pressure on the sensory nerves in the gut wall is felt as pain.

The symptoms of spasmodic colic are those of pain of an intermittent nature. The pain comes on in waves. We have a period in which the animal is quite colicky. Then there is relief for a period of time. Often these symptoms are quite mild, and the attack passes off without treatment. Frequently we see an animal that may be colicky for fifteen or twenty minutes and then, without any treatment, recovers. At first a very active peristalsis takes place and this is usually associated with increased defecation. Later the peristalsis becomes decreased.

Another characteristic of the symptoms is that there is no change in the temperature, pulse rate or respirations as a rule.

The treatment can be approached from several aspects. One is the use, as Dr. Riley mentioned, of sedatives such as demerol or chloral. Other drugs are given to stimulate the sympathetic

nervous system which is inhibitory to the gut, such as adrenalin, or to counteract the parasympathetic by the use of atropine, and some people prefer to use antiferments, or mineral oil and antiferments, on the basis that the whole chain of symptoms has been set off by fermenting food.

Some veterinarians take the opposite approach and will use stimulants of the parasympathetic nervous system which moves the offending material on, to get rid of it. However, the pain is very much intensified by this type of treatment.

If we recognize a condition of stimulation of the vagus nerve creating a syndrome that can be accepted as vagotonia, I think probably we should also recognize the opposite, the stimulation of the sympathetic nervous system producing a condition that might be spoken of as sympathicotonia. There does not appear to be much published on this in large animals but Russell Greig along with other authors have brought attention to this syndrome in horses. Most veterinarians will remember Greig as the man who introduced the use of calcium for the treatment of milk fever in cattle. Greig reported on a disease that is seen in Scotland and Western England and Wales where stimulation of the sympathetic nervous system exists.

It is called grass sickness. In this particular disease one finds an affection of the sympathetic nervous system and yet stimulation of the effector organs occurs. Normally, sympathetic nervous system stimulation results in the secretion at the end-plate of an adrenalin-like substance called sympathin which affects the effector organ. He found that in grass sickness, to quote "You find a disease in which there is little or no fever. The pulse is accelerated. There is a variable amount of thick, glary mucus secreted in the mouth. There is difficulty in swallowing. Regurgitation of ingested food from the stomach may occur. The intestines are in a state of stasis that approaches the absolute. The stomach becomes distended with a very large quantity of fluid."

The stomach may be distended to such a degree that rupture may occur. Greig attributes this to stimulation of the sympathetic nervous system. It produces relaxation of the intestinal wall and it causes very strong contractions of the sphincter at the pylorus, the sphincter at the ileocecal junction and the sphincter of the anus. Fluid accumulates in the stomach since it is constantly being secreted, some is due to swallowed saliva and swallowed air.

It is rather interesting that there have been reports, from time to time, that pylorospasm may be associated with diseases of the liver. Greig points out that in diseases of the liver both tyrosine and phenylethylamine may be released both of which have an adrenalin-like action which may be capable of producing pylorospasms. [Applause]

MODERATOR RILEY: Dr. Wheat will now take the surgical approach in therapy.

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DR. J. D. WHEAT: My remarks are to be confined to the surgical treatment of colics. In trying to present this phase of treatment, I feel like one of the two little boys peeking through a hole in the fence outside a nudist colony. "Wow" he shouted, "there's a bunch of naked people behind this fence." The other boy said, "is that so, what are they men or women"? The first boy said, "I don't know, they haven't got any clothes on."

When considering the question of whether or not to do surgery on a colic case, the first thing we have to ask ourselves, is it absolutely necessary in order to save the life of the horse? If it isn't, don't do it.

When the cause of colic is not definitely established this decision is sometimes difficult to make. There are some conditons that can only be treated by surgery. One of these is intussusception of the small intestine. This usually involves the terminal portion of the ileum. There may or may not be a complete obstruction of the bowel. The last three cases we have had, have been in yearling Thoroughbreds. A typical case history on these three is: Intermittent colicy signs for the past two weeks, slightly elevated temperature, intestinal sounds that are either normal or slightly increased and injected conjunctival membranes. On rectal examination the feces present are coated with mucus, the intestines are moderately distended with gas and pain is evident when the lower portion of the right abdominal cavity is palpated. In some cases a firm painful mass can be palpated. Eventually, these cases will show a terminal acute stage in which the colts symptoms rapidly become worse. If left untreated the horse will die in about two days. On autopsy, the stomach and intestines are greatly distended with fluid and back in the terminal ileum will be found an intussusception.

We have had success with these cases if they are operated upon before peritonitis and shock develop.

A ventral midline incision about eight inches long is sufficient to work through and gives good access to the area. This is a better approach than the flank because often the bowel cannot be pulled up to the incision. A resection of the intestine is not always required. The last colt we operated on had a portion of small intestine telescoped into the cecum by gentle manipulation and traction, the obstruction was reduced.

Another condition that produces acute colic is a strangulated inguinal hernia. This is fatal unless the hernia is promptly reduced. The strangulated portion of bowel is distended with fluid and gas and the mesentery becomes edematous. Reduction of the hernia can be facilitated by asperating the contents of the distended bowel. Instead of using a needle and syringe which soon becomes plugged, I prefer to put a circular purse string suture in the intestinal wall, make a stab wound in the center, force a section of rubber horse catheter through the wounds and tighten the

purse string to hold the tube in place. The bowel can then be evacuated. The puncture wound can easily be sealed by completely closing the purse string as the tube is withdrawn. If the hernia still cannot be reduced, it is necessary to enlarge the inguinal ring in an anterior direction using a blunt pointed bistoury. After the hernia is reduced, the incised inguinal ring is sutured and the hernial sac which forms the external tunic of the testicle is ligated as close to the ring as possible.

Another colic condition is that following prolapse of the rectum. This is a very unfavorable sign. So far, I have had no survivors. The last two have died from necrosis of the rectum as a result of the mesorectum being stripped free from the rectum by the force exerted in the process of prolapsing. I have found no successful way of repairing this condition. If anyone has any suggestions I would be glad to hear of them.

Impactions

Usually respond to less drastic measures. Occasionally an impaction fails to break down with ordinary treatment and by doing a laparotomy the impacted area can be thoroughly massaged and broken down. For the operation, the standing flank approach, using local anesthetic is usually sufficient.

Torsion of the colon is a difficult condition to correct surgically. My success has been one out of three attempts.

The problems in the condition are multiple. First is the large area of bowel involved and the state of shock that the horse rapidly develops. Then, there is the problem of trying to correct the torsion itself. Some cases of torsion are difficult to untangle spread out on the autopsy floor, let alone working blindly in the abdominal cavity. Unfortunately, torsions of the colon accounts for 2.5 to 5% of all colic cases. Colic caused by an enterolith is uncommon, occurring in only one-third of 1% of colic cases. Obstruction of the intestine by an enterolith should make an ideal surgical case. However, such obstructions are apt to occur at the junction of the large and small colon. This part of the bowel is difficult to bring into a laparotomy incision due to its firm attachment to the upper abdominal wall. The result is a poorly exposed field and a contamination of the abdominal cavity.

Strangulation of the intestine is found more frequently in the horse than any other specie of animal. The intestine is usually strangulated by some kind of internal band. These bands consist of such things are acquired diverticula, the pedicle of an enlarged ovary, the ligament between the kidney and spleen, pedunculated lipomas, the spermatic cord of an undescended testicle, and openings in the omentum and mesentery. Because the small intestine is the most mobile part of the intestinal tract, it is the most subject to strangulation.

The severity of the symptoms depends on the length of the intestine involved and the amount of pressure and tension on the

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bowel. Strangulation of the small intestine causes more pain than strangulation of the colon due to its more abundant nerve supply. Diagnosis depends mainly on the findings of rectal examination. If a sudden change from a normally expanded intestine to a collapsed bowel and a tense band-like structure are found, an immediate laparotomy is indicated.

Strangulations are easily found on abdominal palpation through a laparotomy wound and can usually be corrected.

Surgical treatment of colic has not been performed frequently enough to establish any set rules to follow. The available data on the subject is very scarce. Each case must be watched very closely and if surgery seems indicated it must be done early, provided of course you can get the owner's permission to do it at all.

MODERATOR RILEY: Thank you, Dr. Wheat, for a very excellent presentation.

Dr. Adams will now take up obstruction colic.

DR. O. R. ADAMS: I would like to talk about some things that give me trouble and I assume that they are things that give other people trouble. The group of colics that I want to differentiate are thromboembolic colic and the obstruction colics. Obstruction colics include impactions and torsion primarily, although intussusception and volvulus are considered in this group, but are of less importance.

Thromboembolic colic or thromboembolism of the anterior mesenteric artery is a condition that is becoming increasingly more prevalent because of an increased horse population and greater concentration of horses.

I divide thromboembolism into two types: a mild type which survives, and an acute type which does not survive. In the mild case of thromboembolism, the symptoms are a persistent colic that lasts for at least four days or as long as three weeks. The patient doesn't show alarming symptoms. Recumbancy in stall, stretching and lying for prolonged periods on his back are common symptoms. The horse does not usually abuse himself, and does not thrash violently. His pulse may go up to 60 or so, but not to the alarming levels that severe colics do. The mucous membranes look good and are not severely injected.

During the day, he may look quite normal and may eat and drink. The case may appear to be recovered, only to show colic again in a few hours.

The pathology in these mild cases is an obstruction of a small artery, or a partial obstruction of a larger vessel. The wall of the intestine causes pain when the circulation is cut off, and it apparently causes exacerbations and remissions of pain.

In this condition there is very little gas accumulation, whereas in obstruction colics, the pain is from the gaseous distention of the bowel. On rectal examination in the mild case, you will find only a very localized distention of the abdomen, nothing that

requires intervention. Because of the decreased circulation, very often there is atony of the bowel, and feces may not pass for three days, yet there is no gas distention. That is the important differentiation. If you are fortunate, you can actually palpate a branch of the anterior mesenteric artery that shows a thrill. This indicates a decreased amount of circulation through that vessel and is pathological, as far as I am concerned. Fecal examination can be done for numbers of parasites. This is not going to be diagnostic because strongyles are very common. I do not know the significance of it, but we see a number of thromboembolic cases seven to twenty-one days following worming.

In the acute types of thromboembolic colic, where the end result is gangrene of the bowel, or a toxemia resulting from decreased circulation of the bowel, the symptoms are more severe. The animal shows more acute pain, and the symptoms, as far as I am concerned, are not specific. Violent colic symptoms are present as they are in many other colics. Colic symptoms, as far as I am concerned, are not specific for one type of colic. The mucous membranes in this severe case show injection and toxicity. The pulse rate jumps up and approaches 100 or more in the terminal portion of the case. One sign that is significant to me, is that the abdomen does not become tightly distended in thromboembolism. The animal may not be passing feces or gas, and on rectal palpation, very little feces in the terminal portion of bowel may be present. This is usually due to paralysis of the bowel. Two horses, one with terminal thromboembolism and one with terminal obstruction colic, would be very similar, except that gas distention of the abdomen is more severe in obstruction.

In acute thromboembolism, rectal examination may again reveal a partial obstruction of one of the arteries by a thrill in the artery. Tapping the peritoneal cavity and withdrawing a small amount of fluid, if it is present, will be helpful in diagnosis when the bowel is beginning to undergo changes. A discolored fluid with a number of white cells in it on microscopic examination, may indicate gangrene. However, torsion or volvulus with gangrene will give a similar fluid.

The literature on thromboembolism places great weight on the finding of blood in the feces as a diagnostic aid in thromboembolic colic. If I waited to find blood in the feces, I would never diagnose it. We have had many cases of thromboembolism proven at autopsy, and none have shown blood in the feces prior to death.

Impaction is a very important condition to us, and I think its importance varies geographically. We have to do surgery on most impactions. In most cases, we are not successful with medicinal treatment. Most impactions in our area are in Shetland ponies. The diet is usually alfalfa. We feel that alfalfa is very poor horse feed in our area. This is because there are only three cuttings per year and the heavy stems predispose impaction.

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Though alfalfa is fundamentally good from the nutritional standpoint, it is poor from the standpoint of impaction, as far as we are concerned, especially if water intake is low for any reason. In areas where more cuttings per year are grown, the stems are not as apt to cause impaction. It would be very unusual to see a case of impaction on a diet of good native hay. We do see an occasional one on cornstalks or some other type of roughage. We try to advise keeping horses off alfalfa or hold it to a maximum of one-third of the roughage. This is hard to accomplish because it usually runs around $10.00 a ton cheaper than the native hay and the owners do not want to take them off alfalfa because of the economy involved.

Impaction will cause the animal to show typical colic symptoms and the onset is usually very sudden, but the impaction may be several days in developing. The diagnosis is based on rectal examination with actual palpation of the obstruction, dry sticky mucous in the rectum, gas distention of the abdomen, injection of mucous membranes, and an increased pulse rate. Impactions usually are in the transverse colon (the junction of the large and small colon) in our area. Because of the anatomical location, the impactions are difficult to actually palpate and cannot be pulled back into the pelvis to be broken down as they can when they are in the terminal portions of the small colon. Identification of the impaction is all they can usually be done when it is present in the transverse colon. If one can actually get hold of the impaction, and the animal is under epidural anesthesia, many of the small colon impactions can be broken down. The important diagnostic feature in impaction is distention of the abdomen in differentiation from thromboembolism.

Once the pulse rate exceeds 100 the terminal phase of impaction is near. The affected horse may be violent and in the struggle, may break the bowel open. This gives sudden relief and a sudden end, as far as the animal is concerned.

Other types of obstruction colic, such as: Torsion, volvulus, and some cases of intussusception resemble each other very closely clinically, as well as being similar to impaction. Rectal examination would be the main way of differentiation. The distention of the abdomen, toxicity of mucous membranes, the increase in pulse rate, which is very important, all look the same in the obstruction colics. The colic symptoms are not specific for each. I think one can say when an animal has intestinal obstruction, and if an adequate rectal examination can be done, a volvulus or an impaction may be located. But, unless one can actually palpate the pathology, all that can be said is that the animal has occlusion of the lumen of the bowel. I believe one can usually tell the difference between an acute thromboembolism and an obstruction on the basis of the symptoms I have mentioned.

Like everyone else, about the time I believe I am getting

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pretty sharp, a case comes along that fools me completely. This happened to me very recently. I saw an atypical case of thromboembolism which was characterized by high temperature and diarrhea. I had my mind set on another disease. At autopsy, acute thromboembolism was the cause of death. So, I am not saying that in all cases an accurate diagnosis can be made.

To summarize the difference between the different types of obstruction colics, the only way one can identify the different types is by actual rectal palpation, or doing a laparotomy. We do approximately 13 impaction operations a year, mostly on Shetlands. Of course, as Dr. Wheat indicated, you do not get much chance at torsion. I will show you a movie of one successful torsion of the small colon, and one successful intestinal anastomosis in a case of strangulated scrotal hernia. I will show you two surgical approaches for the impaction operation.

As far as enterolith is concerned, as a cause of obstruction mentioned by Dr. Wheat, I will show you the picture of one from an English jumping horse, which I diagnosed as impaction because I could palpate the mass in the transverse colon. These are quite rare. It really makes no difference as the treatment is surgery in both. It was removed by laparotomy and the horse lived.

Gastric dilation is one of the things you do not see very often, but we lost three cases in a week. Some of the uncommon conditions go by streaks. I think that whenever a horse shows vomitus in the nostrils, one should be suspicious of gastric dilation. Watch the esophagus. If the horse tries to vomit but can't, peristaltic waves in the left side of the neck are visible. The head is carried so that the nose is near the ground. Most cases of gastric dilation die. One died with a stomach tube in place at our clinic. We were aspirating the fluid as best we could. All three died with ruptured stomach. One, in addition, had a ruptured diaphragm.

Spasm of the pylorus is present in gastric dilation and is a difficult thing to relieve. We cannot relieve it with ordinary drugs. Our pharmacologist tells us that Reserpine possibly would help.

Now we will go to the movie and then a few slides.

This film shows one method of correcting impaction. This is the midline incision, the animal in dorsal recumbency.

We do not try to obtain surgical anesthesia on these patients. They are so toxic that ordinary doses of anethesia would kill them. They will die before surgery can be done if deep anesthesia is attempted. We give them tranquilizers and no general anesthetic if possible. Local infiltration anesthesia around the operative area is then done. A minimal amount of general anesthesia is used in this way. Actually, our record on these is very good. We have lost only one following surgery, and that was from shock in a Shetland. We have lost several prior to surgery from anesthesia. That is why we are careful.

This shows the area of impaction and how small it can

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actually be in a Shetland. It is as hard as a rock and about the size of a turkey egg. It was gently massaged and broken down. One very rarely must actually do an enterotomy and open the bowel. It may take some time. Blunt pressure must be used.

This is the standing flank incision. The horse is under Sparine and local infiltration. The fascia of the oblique muscles of the horse do not have good holding power as they do in a cow, and they will tear when sutured. We use a grid incision through the muscle and break through the peritoneum rather than a vertical incision. When suturing the grid incision, the internal and external oblique muscles practically fall together.

In the standing flank operation, there is a risk of inability to bring the impaction out. In that case, go ahead and try to break it down inside. On this one, we were successful in bringing it out.

This shows the small colon here, through a left flank incision. The animal's head is facing to the left. The impaction is being broken down. We have tried all forms of drug therapy and have had poor results in breaking down impactions by their use. If one is going to surgery, it must be done reasonably soon to get good results. We anticipate that in many cases we are not going to be able to correct the condition medicinally.

Now if we could have the slides, please.

[Slide] This shows the aorta and the anterior mesenteric artery. This is a typical thrombus and aneurysm. The aneurysm is the distended portion and the thrombus is the reddish portion at the center. When the thrombus is crumbly in nature, many thrombi break off and may occlude as many as twenty of the vessels to the intestine. The anterior mesenteric artery furnishes blood from the middle of the small intestine to the middle of the small colon. As a result, lesions can be present in many areas.

[Slide] This is an enterolith cut in half. The white paper point in the center shows a nail that started the whole thing. I assume this stayed in the large colon for a long period of time. This horse had a history of developing diarrhea at the Royal Show in Kenya, and then showed symptoms of impaction. I palpated the mass rectally but could not get a good grip to tell it was so hard. It turned out to be about 4¼ inches in diameter, and weighed a little over a pound. This animal survived enterotomy, and the surgery was done on the farm.

[Slide] Here is intussusception caused by ascarids. Here is the intussusception portion here. Ascarids may account for some of the intussusception cases. Dr. Wheat mentioned most of intussusceptions are seen in younger animals, and ascarids are more common in the younger animals.

Thank you.

MODERATOR RILEY: We will now entertain questions from the floor.

DR. J. M. BREWER [West Chester, Pa.]: I would like Dr. Adams to talk about the treatment of the milder form of thromboembolic colic.

DR. ADAMS: This is a real good question. What are you going to do? I probably should start by saying I don't know. There was a report out of South Africa saying sodium nitrite was good for distention of the arteries, and so on, in thromboembolism. I tried it on two cases, with fatal results, not from the drug but they both died, and were confirmed on autopsy. I used this product called thionitrite that we use for HNC poisoning. I think on the next ones I will try intravenous verasene. I think I will use 250,000 units of that twice a day and see if it has any dissolving effect. I do not know why I did not think of that. Somebody brought that to my attention that it was a drug for that type of thing.

DR. C. H. REID [Hollywood, Calif.]: Nothing has been said here about impaction of the cecum. How often do you see that and what do you do for the symptoms?

DR. ADAMS: As far as impaction of the cecum is concerned, I would have to limit it to this, to say I have never seen impaction of the cecum except as secondary to impaction more posterior to that, where the material has been backed up into the cecum, and it is also packed. The literature talks about it like at least at one time it was quite common, but I have no experience with it. As soon as I finish, maybe some of the other panel would like to answer that.

DR. WHEAT: With regard to cecal impaction, I have seen one case. That animal developed diarrhea with the condition. The impacted area of the cecum was by-passed, and eventually the impaction did not break down. The cecum necrosed, and the animal died. There was still material coming through the animal in the form of diarrhea, where it by-passed.

DR. J. C. O'DEA [Avon, N. Y.]: In the hunter and jumper division, where they plait the horse's tail, within an hour before a class you will get a call to hurry up. The horse will be down and sweating and rolling. Everybody is sure it is colic. In about nine out of the ten cases, if you untie the tail that has been plaited in such a manner that it has made the horse very uncomfortable, he will get over the so-called colic. At the Pan American Games we had a few cases of "colic" that were cured in about three minutes.

DR. H. M. SMITH [Hanover, N. J.]: I wonder if the panel would care to express their feelings on the incidence of colic which we encounter in shipped horses, and what their recommendations would be as a preventive measure prior to shipping.

DR. KEOWN: I think that the incidence of colic in shipping is largely due to this autonomic nervous system phenomenon, as I mentioned before. The horse is extremely nervous, and there is an increased amount of adrenalin released in the body which

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causes, for a period of time, a stasis of the musculature and a contriction of the sphincters. Then the material that is eaten begins to ferment, causing irritating materials, and he gets that reflex stimulation of the parasympathetic.

I think that is also the explanation for the intermittency of the attacks, that for a while there is parasympathetic, and then, with the secretion of acetylcholine at the nerve endings and stimulation of the musculature later on, there is stimulation of the sympathetic. So, we have an intermittency. At least that is the theory we are working on.

DR. SMITH: Is there any recommendation for preventive measures?

DR. KEOWN: I would say reduction of the feed at the early part of the shipment. There is no doubt about it that you will get an increased amount of colic if the animal is fully fed and subjected to emotional stress. For example, if you do any surgery on an animal, even in standing surgery, you get a higher incidence of colic in animals that have not had the feed reduced, because there is a stasis and fermentation, and very often a lot of gas.

DR. ADAMS: Another answer to the question on shipment of horses and prevention of colic, the use of tranquilizers is quite good in slowing down and preventing some of the cases that get colic in trailer trains, especially.

DR. WHEAT: Another thing I think that is overlooked on shipping these horses is to give them a dose of mineral oil before they are loaded out. These horses oftentimes are short of water during the shipping. If you can give them a mild amount of oil, it will certainly cut down the incidence of colics from vanning.

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VETERINARY ASSISTANCE TO FOALING MARES

ARTHUR H. DAVIDSON, D.V.M.

In accepting a place on the program of this meeting to discuss some facet of brood mare treatment or care, it occurred to me that perhaps no one has discussed foaling problems. As a consequence, I will attempt to discuss, first of all, dystocias in mares. I realize that many of the statements I will make are incomplete and need further explanation or comment and, if you would like to interrupt at any time, I will welcome it.

To the best of my knowledge, there has been very little literature published on this subject in recent years. However, the old classic texts are good, and the mares certainly have not changed. In general, everything is the same except many of us do not have adequate contact with horses during our youth or during school years. In other words, a newly graduated veterinarian may not have seen a mare foal and, consequently is not prepared to do what is necessary.

I might say that the equipment that I have always carried in my car—it rides year after year—usually consists of two or three O.B. chains and handles to match, and a fairly long snare, and a Fetatome. The particular kind I have is a double-barreled one to which the tap screws in the end of the stylets. I carry two sets of these taps. One set is attached, on a short wire, braided wire, and the other set of taps on a longer wire so that I can fit them to whatever part of the fetus required. Also I like to have a tracheal tube, which I will explain later.

When called to a foaling case, it is desirable to waste no time, for a few minutes can ruin a mare or make a difficult case.

Upon arrival, try to find out how long the mare has been in active labor or how long she has been down. Quite often the mare will be down and straining desperately. Try to get her on her feet for the examination. It is much easier with the mare standing.

Of course, the tail should be bandaged, and the mare washed up. The examination should be as complete as possible but done rapidly. All parts of the foal that can be palpated should be identified. A decision of what must be done is then made.

The majority of dystocias are relatively simple, with one leg or two, or the head retained. Early in labor it is easy to repel the foal enough to correct the position. However, after the uterus has contracted and the foal is jammed into the pelvis, it becomes a difficult situation. After fluids are lost, it may be necessary to balloon the uterus slightly with a lubricant, before a start can even be made. The mare may be down and resisting every move with

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violent straining. Sedation or anesthesia then becomes necessary, before you can work.

If straining cannot be controlled and anesthesia is not wanted, the use of a tracheal tube will save the operator's arms considerably. I was told that trick by a predecessor, and I don't know that it is of general knowledge. You can insert a tracheal tube in a mare or, I suppose, in a cow, and she cannot hurt your arms. It has made working considerably easier at times.

If there is a leg retained and there is room to do it, repel the foal enough to raise the foot. Always cover the foot with the hand to prevent lacerations.

If the foot cannot be brought out, it may be necessary to attach a chain above the knee and take the foal with the leg folded. If ankylosed at the knee, it is best to amputate the leg at the knee.

Sometimes both front legs will be retained and folded back. Chains should be attached to both legs as near the foot as possible. The traction on the chains must be applied upward. I sometimes use my shoulder as a fulcrum and have the chains up over, so the traction from back of me pulls the foot up. If labor has progressed too far, it may not be possible to bring the feet up. Amputation is then indicated.

Frequently the head and neck are presented and completely fill the pelvis so that the operator cannot work. If one foot is retained, it is likely out of reach. If both are retained, it is still worse. Usually it will be necessary to remove the head and as much of the neck as it possible. If the legs cannot be brought up then they should be amputated.

It is very hard to take both front legs while flexed at the knees. In other words, in some cases you might have the front legs retained, fcaled at the knees, and you haven't enough room with both knees, it is hard to get them into the pelvic cavity and to extend the shoulders enough to deliver them. You probably have to amputate.

Many times, all that stops the process of foaling is that one or both shoulders are not extended, and the elbow of the foal is jammed against the pelvis. Knowing how, when and what part needs traction may be all that is necessary.

When labor starts, the front feet may push up at a sharp angle and tend to come through the roof of the vagina and into the rectum. The assistant must prevent this by protecting with hands and pulling the legs out past the vulva. A head may do the same thing. Every year several mares are injured in this way; maiden mares are particularly susceptible to this.

When the foal's head is turned, uterine contractions will displace it backward, either toward the hips or down under the foal's belly. Sometimes it is nearly impossible to reach the jaw to attach it. The use of a snare with stiff wire is helpful. Chains are very

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hard to manage with the finger tips, at arm's length. If the head is turned and lying over the foal's back, it is usually necessary to repel the foal while assistants apply traction. In this way you get a little slack as the foal has to be repelled before you can snap the kink out of its neck and straighten the head. If the cervical vertebrae are fused, it will be expedient to amputate as near the base of the neck as possible.

If the foal is alive and normal at first examination but the head is in a malposition so that chains or snare are used, one should be careful not to injure the jaw any more than absolutely necessary. It will, however, stand considerable abuse.

Deformities must be handled in any way suitable. Enlarged cranial cavities, hydrocephalus, are occasionally encountered. Some are so large they will not even enter the pelvis. Usually these heads are soft and may be reduced in size by draining and crushing.

Barrel-chested foals minus front legs may be encountered and are a real problem. Desperate application of traction, regardless of damage to the mare, will probably be the answer. I have run into three or four of these, with just a stub of a front leg but a tremendous barrel chest. The foal was coming straight, and the attendants had already labored a considerable time, trying to deliver it. It is in such a state that all you can do is proceed as they have started, regardless of what happens to the mare. If seen in time, however, and recognized, it probably is possible to dissect the chest and sever some ribs or xiphoid cartilage and reduce the circumference of the chest cavity.

Nearly every spring you may encounter what appears to be a normal foaling act but the foal stops when only half delivered. On examination, the hind feet are found under the belly of the foal and the hocks braced against the mare's pelvis. Occasionally there may be room enough to attach chains to the hind feet and then to extend them up under the foal. When this is not possible, the only chance you have is to sever the trunk of the foal, repel the hips while using traction on the hind feet of the foal, and it can be delivered in this manner.

After the posterior stump has been turned, it still might be possible that you will have to split the pelvis and the lumbar region in order to deliver it. That happened to me once.

True breech presentations are very difficult to handle in mares. The foal's legs are so long that, when encountered, the foal cannot be repelled enough to bring the legs up. Very early in labor it might be done but after the mare has been in labor for sometime it then becomes necessary to remove the hind legs at or above the hocks. Enough stump must be left to which to attach chains. With shortened legs, the position of the foal can then be corrected and delivery accomplished.

Quite often a large foal will be presented one-quarter or one-half rotated on its long axis. A quarter rotation will correct it-

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self, if the mare will get up and down a few times. Assistance can be given by crossing the legs and rotating the foal as the mare labors.

A half rotation or upside-down foal may be turned if it has not been pushed into the pelvis too far. If delivery has progressed, it may be necessary to bring the foal out as it comes. The direction of the pull or traction must be dorsal to the mare.

Direction of traction must be calculated in all deliveries, both normal and abnormal. Traction in the wrong direction accomplishes only damage to the mare.

To summarize some of my thoughts on this subject:

It is easier to make examinations of the foal with the mare on her feet.

It is much safer to know where all the feet are and the size and position of the foal before delivery is attempted.

If the mare is down and has been in labor for a considerable length of time, she may not get up. Sedation and lubricants may be used before an examination is possible.

Asepsis is nearly impossible but cleanliness is necessary in foaling mares.

Gloves may be used but in long and strenuous procedures they add to fatigue and are usually torn to shreds.

If repulsion and dissection of the foal is necessary, it is usually advisable to anesthetize the mare. Her help is lost in delivery after correction is made, but constant straining will cause unnecessary damage to her. A mare in labor can absorb unusually large amounts of anesthesia.

If possible, work inside the fetal membranes as they will protect the mare and lubricate the foal during delivery.

Damaged mares cannot always be treated immediately. Suturing tears of the vulva must be left until the next day, or later. Lacerations within the vagina should be observed and treated as needed, until healed. I have a tendency to under-treat rather than over-treat. Cervical lacerations may look terrible the week following injury but if the muscles are not severed, they will usually heal and be serviceable.

If the uterus is not damaged, it will contract and clean itself.

I thought I would talk a minute about the approach to normal foaling cases, as I see it. Many times one is called to a foaling case that resolves itself into a normal delivery.

There are some things that should be done and some things not to do with a mare foaling.

After examination and you are certain everything is normal, it is best to observe the mare's behavior for a few minutes. Some mares will break out and be uncomfortable for quite some time before they start to move the foal. If she lies down and starts applying pressure, you usually assist to some degree. Check head and front legs, making sure that they are coming straight, and

then pull a little on the front legs. Hold a little pull, perhaps 50 or 75 pounds, as the mare strains. If things move along gradually, all well and good. It is wrong to try to bring the foal too rapidly. I do not like them to hang with pressure on the chest cavity very long, however.

When pulling the front legs through the pelvis, the force of the pull is straight back. After the chest is delivered, direct the pull somewhat downward. When the lumbar region appears, the pull should be directly down so the stifles of the foal will clear the pelvis.

After the foal is delivered, let it lie for several minutes before breaking the umbilical cord. Sometimes the mare will move and break it herself, which is all right. When you must break it, try to do it between two pads of cotton or gauze soaked in iodine, metaphen, zephiran, or something that is a fairly efficient antiseptic. If there is hemorrhage from the cord it may be tied, although it is seldom necessary. Tough cords can be incised or snipped with scissors. These may, however, be infected cords and will be troublesome later. A big, thick hemorrhagic cord quite often is an indication of an infected placenta, and the foal may show evidence of it in a few hours.

Usually a mare will lie down for a few minutes after foaling. This is good, but always check her pulse in about fifteen minutes. If she stays down for some time, slide the foal to her head where she can see it and not be worried about it.

Most foals get up, after much falling around, in thirty to sixty minutes. Some brood-mare men assist them to nurse and others only encourage them slightly. Some foals are a real problem, however, and do not learn to nurse for many hours or days.

There are some mares who will not lie down to foal. The pain seems to discourage them. These mares are very hard to foal and it requires a good deal of traction and a good deal of agility to aid them. Sedatives might put her down, but if they are used you will have a drugged foal to nurse.

There are many situations on this subject and bordering subjects that could be discussed. However, if time is available and you wish it, I would welcome any questions you might have, or questions on something I have said. I would be very, very glad to hear from you. [Applause]

CHAIRMAN WOODCOCK: Thank you very much, Dr. Davidson.

Are there any questions from the floor?

DR. C. H. BURGER [Bakersfield, Calif.]: Do you encounter many torsions in the uterus, and how do you handle them?

DR. DAVIDSON: In my experience, I have never run into one of them in a mare.

DR. HORTON K. HOWARD [Saratoga Springs, N. Y.]: What about retained placenta?

DR. DAVIDSON: I expect you could write quite a book on retained placentas. I think the same is true with mares as is true with cattle, if I remember cattle practice correctly. If the brood mare is in good physical condition and does not happen to pick up an infection during gestation period, she is very apt not to have a retained placenta. However, if you do, we encourage contractions of the uterus first. As I remember, we used to let them go for about twelve hours and then take them manually. I have stretched this out to 24 or more, if the mare is all right. I do not object to manually taking them, but I feel I do some damage. If the mare is not worried about it and is not toxic, not running any temperature, and is doing fine, I will let it stay for a while. We used to use such things as posterior pituitary extract to contract the uterus, which is valuable within the first few hours. They have other things now that are still better. We do not have too much of it, but there are a few. However, I would wait fifteen hours or more, if everything is all right, before I attacked it.

DR. D. L. PROCTOR [Lexington, Ky.]: I would like to make a comment on the use of agents, chemical agents, to cause contraction of the uterus. I, personally, sincerely doubt that the aid to contraction is worth the potential damage that might occur. I suppose more mares over fifteen years of age die of postpartum hemorrhage than any other single thing. Nearly all of our agents that produce uterine contraction raise blood pressure and potentially can cause postpartum hemorrhage.

I think that the agents used for contraction of the uterus should be used with a great deal of care and thought.

DR. DAVIDSON: You are probably right. Of course, physicians do use them a great deal but the uterine arteries involved are not as heavy. You may have a good thought, Dr. Proctor.

DR. J. C. O'DEA [Avon, N. Y.]: The other observation which rules them out is the fact that if posterior pituitary is used shortly after foaling, involution of the uterus and the chance of getting that mare right back to breeding in nine days is much less than if you let nature and the normal flow of pituitary take over.

DR. DAVIDSON: I expect you are right, that, when you attempt something, you usually cause a reaction. However, it is quite a general policy in Kentucky that any mare having a retained placenta for several hours, has her breeding automatically postponed.

DR. E. H. WILLIAMS [Houston Texas]: Doctor, what are your views on epidural anesthesia in foaling mares?

DR. DAVIDSON: Somewhere way back along the line I became afraid of it and have never used it. I have seen it used; maybe I have used it a few times. I do know that a foaling mare is easy to panic. If they get unsteady on their feet, the hind legs become partially useless to them, they will fight it, and you have

a terrible time. Maybe things have improved, but I was taught not to use epidural in mares unless you had to. I have never used it routinely, or seldom, if ever.

I think the last time I used it was in docking tails of colts in the Midwest years ago. It can be used. I do not think it is as efficient as it is in cattle, not nearly so.

CHAIRMAN WOODCOCK: Any other questions?

Thank you very much, Dr. Davidson.

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ENCEPHALOMYELITIS – A BRIEF REVIEW OF POSSIBLE ECOLOGICAL FACTORS INVOLVED

OSCAR SUSSMAN, D.V.M.

It is really a pleasure to be here. I am sincerely appreciative of being at a meeting where so many of the men stay at the meeting to hear the papers. It is so different than what usually occurs.

In speaking of encephalitis, I would like to suggest that perhaps — and I am sure Dr. Kester agrees with me because we had lunch together — we might do well, as a group to cease using the term "equine encephalitis" or "equine encephalomelitis."

Up until 1938 it was considered that this was a horse disease, and it was called "Equine Encephalomyelitis-Eastern variety." It was found that the Western variety and St. Louis and Venezuelan, and a number of others were in the same general group.

I think, as we went further into the epidemiology of the disease, we began to find that, really, it was somewhat of a misnomer, and that it does cause an encephalitis, and in horses it does cause an encephalomyelitis. If it were found in a horse it could be called equine encephalomyelitis, and if it were of the eastern variety, you would not be incorrect to call it eastern equine encephalomyelitis.

However, what do you call this same disease if it happens in a sparrow or blackbird, or in a thrush or egret or if it happens in a pheasant or happens in humans?

I think, perhaps, we might get away from the onus, as far as the horse is concerned, if we call it eastern encephalitis, if we did sort of shy away, and rightfully so, from calling it "equine" because it gives a bad connotation to the horse, in so far as the spread of the disease is concerned.

Before I go any further, if I may, with your permission, I would like to review something that I am sure most of you know but, perhaps, you do not really have at your finger tips, if you are not in this end of the work. That is, when you see the figure "10" and then you see "-5" and then you see "LD_{50}" I am not sure that we all know what it is unless we go back and start to think a little bit. This means quite a lot, as far as the spread of a disease is concerned, when we are talking about a variety of species.

"10^{-1}" really means this: If you took some blood, the serum from an animal or something, and diluted it 1 into 10 parts of sterile diluent, and then took that amount of material and in-

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jected it intercerebrally into mice, you could then say, if that material killed 50 per cent of your mice, that you had a 10^{-1} - LD_{50} intercerebral dose of mouse virus.

If you then go ahead and dilute that material one more time, you then have 10^{-2}, which means you have actually brought it down from the original dilution of 1 into 10. Then you took a part, one-tenth, of your first dilution and bring it into the next tube. You then have a dilution of 1:100. That is 10^{-2}. 10^{-3} is when you take one-tenth of that second tube and put it in a third tube. Then you have 10^{-3} or 1:1000.

So, you start with 9 cc., as an example, and have a series of tubes. You take out 1 cc. and put it into your first tube. You have 10 cc. of solution—10^{-1}. Take 1 cc. of that, put it in the next one, and you have 10^{-2}, and so on down.

This is relatively important in thinking about encephalitis. I might tell you that most of the work that I am quoting is from Dr. Kissling, Dr. Stamm and Dr. Holden. While I am telling you that, I might say if you would like to get a book that would really be of some assistance to you for a review, you might get a book called "Animal Disease and Human Health" published by the New York Academy of Science. It has a wonderful review of all of the encephalitides in the front of the book.

The things that have been found out are that herons and pheasants usually carry a virus titer of 10^{-3} - LD_{50}; that is 1:1000 dilution of the serum from pheasants and herons at the time that they are infected will kill 50 percent of the mice into which they are injected, and that herons and pheasants go up to 10^{-5}. Whereas small birds, such as grackles and cardinals and sparrows and pigeons have a dilution of 10^{-6}. That means, if you dilute the serum from a sparrow or pigeons six times, into six tubes, you can take the sixth tube, inject it into mice and kill 50 percent of the mice, if the virus were present. That means that they have more virus floating in the blood stream than is present in the larger birds such as herons, egrets and pheasants. So, you have more of a danger inherent in taking blood either directly or indirectly from a small bird, these passerine birds, and the pigeons and the sparrows, and getting transmission than you do from the larger birds.

Now we come to the horse. It has been found that the horse has $10^{-0.5}$, as high as 10^{-5}, but that in most horses the maximum amount of titer is 10^{-3}, meaning that, if you took the blood from a horse that was sick with encephalitis and had virus floating in its body, you could go three tubes down, and with that you could infect half of your mice. But, when you went to the fourth tube, you would not. Whereas, to go back to the pigeons, you would find that you could go down as low as 9 dilutions and still infect half of your mice.

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Now, then, one more factor is needed. You do not get the blood directly from the pheasant, from the horse or from the pigeon. So, from what we now know, you need a vector. We have assumed, on the basis of what we know, that the vector is a mosquito.

Dr. Kissling did some work in which he found that mosquitoes need a titer of 10^{-4} to 10^{-5} to become infected, most of the time. So that, if mosquitoes are biting an animal that has a virus, in the blood stream, with a dilution of 10^{-3}, the mosquito does not become infected. In only one case, so far as we know, and that was Kissling's work, experimentally, has ever shown that horses infect mosquitoes. In this case they took virus, injected a horse, and then allowed certain mosquitoes to bite the horse. It was only in this one case that they were able to prove the mosquitoes were infected. This horse, of all the horses examined previously, had the highest virus titration in its blood stream. So that, normally, a horse does not have enough virus titrated in its blood to infect mosquitoes. We do not know enough about humans to know, but we do know that from most scientists' viewpoint, horses and humans are in the same general category.

This bring us back to what Dr. Woodcock was saying, that you can have hysteria or horseteria, depending upon what is done by control agencies. Unfortunately, we found in New Jersey that people became frightened of horses. Horses were to be moved to the Garden State track, numbers of people called our department saying they did not want them.

Really, if we look at this thing in this light and we start thinking about the ecology of the disease, there are several things we have to answer, which we have not answered. Let's just think. In Louisiana there were 14,000 horses in 1947 that were infected with this disease, and in New Jersey last year there were 20 people. This resulted in a great strain on the racing industry in our area; it could have been much more.

Therefore, I say we should evaluate what we call this disease. Some of the questions we have to answer are: Should we restrict the movement of horses during the presence of the disease? If so, what do we mean by "the presence of the disease"? Does it mean if someone who is collecting birds finds it in some wild birds, that is enough for restricting the movement of horses or humans? Because, you see, they did not stop humans from going to New York and betting money from New Jersey, but they did try to stop horses from going there to collect any of the money. We felt rather badly about it because our state is dependent upon the tax money that we get from the tracks. That actually is one of the things that, scientifically, I think, has to be answered, and that is we do not really know the potential of what humans do. We do not know how many nonclinical cases of humans there

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are that actually have the virus and how much of a virus titer they have.

So, if we find the disease in mosquitoes or in wild birds or humans or in horses, which one of these is evidence enough for stopping the movement of either those species or any other species? If the evidence of disease is wild birds, then we must immediately consider that in New Jersey and on the East Coast there has consistently been found, as early as July, virus present in wild birds. This would mean that no movement should occur at least of wild birds. In this case, being facetious, you would have to do what England tried to do, stick up balloons and nets and try to keep the wild birds from passing.

I do not think that any of you are unaware of this fact, that this disease occurs normally when there is hot, wet, humid weather, and this also is during the mosquito season. But we do not really know, as happened in some other diseases, whether or not there is a certain time element of the virus itself that just works at that time, regardless of whether it is the hot, wet, humid season, but more a matter of the time factor. So that actually you would have the virus present, and you need mosquitoes or you needed the weather to make mosquitoes want to bite. This may seem a little far-fetched, but normally Culicidae melinuria, which is one of the mosquitoes that bites and has been infected, normally does not bite humans.

If you had a change, so they got a drier season, after they were just being produced in the marshes, and there was a wind, you will find some mosquitoes changed their biting habits. We do not know enough about what changes the biting habits in order to determine when certain mosquitoes, which normally do not bite humans, which may bite birds and not normally bite horses change and begin to bite horses and humans. Confusing? I'm sorry; it is confusing. That is, normally, a mosquito will bite a human, if it is that type of mosquito. If you change the ecology, if you change the picture of the land and the moisture and the time, in some respects, you will find that these mosquitoes then start to bite other species of animals. That is what we do not really know at the moment is happening on the East Coast. I am telling you what we do not know. I told you before several of the things we do know. That is, horses do not normally have enough virus in them to give us the trouble of which they are being accused.

In horses, we know that the duration of the viremia is very short, normally, less than four hours and, at the maximum, no more than 24 to 48 hours, whereas in birds the viremia may be as long 3 to 4 days.

One other thing that I would like to bring in for your confusion, if you want to put it that way, is that we have evidence now that there is an increase in birds, and you probably are

aware of this. You find in grasslands there are approximately 144 birds per hundred acres, and in forests you know that there is a considerably larger number of birds, and you have about 450 per hundred acres. Whereas where you normally think there would be less birds and less species of the wild type, including insects, where man has made a forest edging, (that is where a road has been cut through a forest, and where there is edging), like the Garden State Parkway, or Route 9, where we had our trouble in New Jersey, you find that there are as many as 800 to 2,000 wild birds per hundred acres.

This means, going back to the other part of what we know, that where you have wild birds that can carry the virus, you are going to have a great many more wild birds present in closer proximity to humans that are possibly carrying this virus, and can therefore act as the carrying agent to the human being.

In that situation what you really need, then, is to remove your susceptible population. In your case, most of you are interested in horses.

Some work has been done by Dr. Holden which indicates you can have bird to bird transmission. That is, in caged pheasants, we have found that you can have one pheasant that is infected, and no mosquitoes, and the disease will spread in the pen. That means that by feather picking or some other aerosol transmission, it can spread within a pen.

This has not been found true among horses. We have yet to find any horses that could spread the disease from one horse to another. Also, we have not found any human being that has been able to be identified as having this disease transmitted to him from a horse. We know of no case of that type. That is exclusive of laboratory workers that are handling virus that came from an animal.

In conclusion, I would like to point out that you have several things you have to think about as veterinarians associated with the race track industry and horses. First, that is you can remove the susceptible population by properly immunizing each horse. The question as to how often you have to vaccinate I think still has to be determined. But, in the light of our present knowledge, I would say that, at the minimum, you would have to vaccinate at least once a year, and some of the men in the field believe that it should be done every six months, with a booster dose.

The second thing is, around your race tracks, you should attempt, in the light, again, of our present knowledge, to have a control made on the mosquito population, because we do know that the mosquito population is indicted in the spread of this disease, at the moment. They may not be the sole spread. They may not be the mechanism by which the disease stays in a territory, but we do know they do have virus and can transmit it, and they should not be around horse stables or race tracks.

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The third thing, and many of you may have done this before, realize that sparrows and pigeons around barns nowadays, in the light of what you now know, are not very healthy type pests. So, act in a manner to remove the sparrows and pigeons from near your race tracks. [Applause]

PRESIDENT KESTER: It is not just race tracks that are involved. We owe a service to our communities in general. An outbreak of encephalomyelitis could mean financial ruin for a race track, just because of misguided public hysteria.

There were several summer resorts that likewise were almost financially ruined because there were a few horses in the community. The horse was what the people were afraid of. They had no idea where the trouble was.

Did I catch this portion of it straight, Oscar: So far as we know, there has never been a human case where the horse has been suspect as the origin of the virus?

DR. SUSSMAN: That is right.

PRESIDENT KESTER: There has been one case where the disease has been transmitted by the mosquito from one horse to another horse, that is the experimental case of Kissling. This horse, the original horse, was artificially inoculated. Is that right?

DR. SUSSMAN: Right.

PRESIDENT KESTER. And his titer was higher than we normally find in the field, on a horse.

DR. SUSSMAN: His titer was 10^{-5}, and ordinarily it is down as low as 10^{-3}.

PRESIDENT KESTER: In nature we have never found a horse with that high titer.

DR. SUSSMAN: That is right.

PRESIDENT KESTER: The attempt to transmit the disease from the No. 2 horse to the No. 3 horse was not successful, which again simulated field conditions.

DR. SUSSMAN: Right.

PRESIDENT KESTER: I think it is important to bear that in mind. You frequently hear the statement tossed around that you can transmit the disease from one horse to another. This is how it was done.

That leads us up to the question of what does the vaccination protect. It protects nothing except the horse that is immunized. The horse is not a source. If I am correct in my deductions, your vaccination protects nothing except the horse you immunize.

DR. SUSSMAN: It protects one other thing—horseteria. If every horse were vaccinated in an area where the disease is present, it would be very difficult for anyone to get hysterical about horses. They would presumably not be able to get the disease.

My personal reaction would be that it would be much more

efficacious, from the standpoint of our position, if all horses were vaccinated.

PRESIDENT KESTER: I think that is a point well taken, and it should be recognized for what it is. It is not a movement to protect the public health; it is a movement, on the part of racing, to vaccinate the horses to protect their own investment, so they won't, by chance, have an infected horse at the track. It is that simple.

DR. SUSSMAN: Right. I think it would be silly to try to do it at the tracks. I think, if it were going to be done, it should be done, certainly, before the racing season started, and it should not be done in the atmosphere of "If you don't vaccinate them, everyone is going to die." I think it should be done as a preventive a long time before.

PRESIDENT KESTER: In other words, you feel a client should be encouraged to vaccinate 1) to protect the host and 2) to protect against the chance at the race track that he might have a sick horse.

PRESIDENT KESTER: I think the more we emphasize that to our client, the more we can do for the client and the horse. Your Executive Committee has been kicking this around. We are developing a resolution to be presented to the AVMA and used elsewhere the best it can, to get this name changed. It probably is an impossible task. Maybe it will serve to disseminate a little factual information in getting the horse off the hook.

CHAIRMAN WOODCOCK: Thank you, Dr. Sussman. [Applause]

DR. CHARLES H. REID [Hollywood, Calif.]: I would like to ask Dr. Sussman if he has done anything as far as wild animals, predatory animals, mountain lion and such as that.

DR. SUSSMAN: As far as wild animals are concerned, there has been a definite neglect in trying to find mammals that were infected with this disease, that might be carriers.

In New Jersey, for example, we are attempting to collect wild rodents and other mammals that might carry it in our state. There has been nothing done to control wild animals. There has not been enough evidence to indicate there was a need for control.

DR. JAY CLAYTON HANSEN [Beaumont, Calif.]: There is one other problem that I think might be considered in the vaccination program, and that is to my knowledge at least, we have at the present time vaccination for the Eastern and Western strains of virus. Most of the Southwest use the bivalent vaccine. What are we going to do with the St. Louis and some of the other strains present? I think there are five listed in the literature.

DR. SUSSMAN: There may be need to have a trivalent strain at some time. In New Jersey we have both the Eastern and

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Western virus because Dr. Holden recovered Western and Eastern virus from in wild birds.

DR. SUSSMAN: One point I did not mention is this: In New Jersey we had 40-some-odd horses clinically diagnosed by veterinarians as having the disease. This is no reflection, again, on the veterinarians concerned. But out of the group we were able to get approximately 25 heads and got them rather quickly, that is the brains. We brought them into the laboratory. I think we only recovered virus from nine of them, and about eight showed encephalomyelitis under pathological slides. That would be 15 out of 25. I don't care how you cut it, it is only 15 out of 25 that, unless you are taking a wild guess, died with encephalitis.

If one doctor sees a horse drop and it has encephalitis, everyone has encephalitis. There are many causes for encephalitis. It could be toxic encephalitis, chemical encephalitis. It could be a number of things. Even if it were only them you should find the encephalitic lesions in the brain. In some of these we did not find the encephalitic lesions in the brain. The horse died; it had some symptoms possibly recognized by the veterinarian, that looked like encephalitis, and it is fairly definite what they are. But even out of that group, when we had men quite well trained in this work, we still had that percentage that did not have the disease.

DR. CHARLES H. BURGER [Bakersfield, Calif.]: I would like to comment on this encephalitis picture. I think, if you have seen it clinically and followed it with blood studies, involving a rise in titer, you do not have too much trouble diagnosing it. I have diagnosed as high as 8 cases in a season. Clinically diagnosed 16, proved 8 on blood test. The other ones, for various reasons, the horses were shipped out and I could not get a second sample.

I think veterinarians, in an area where you have a lot of encephalitis, become quite efficient in diagnosing clinically. It is always wise to prove it with blood samples.

DR. SUSSMAN: I agree. In the case we are in, I think it is very important that you realize that, if a horse dies, you should get as quickly as possible a piece of the brain tissue and in as sterile condition as you can. It would be wise to get as quickly as possible, if you do not have a horse dying, a blood sample, and keep that sample not frozen in an ice-box. Then ten days later get another sample, and then both of these samples be submitted at the same time.

A single sample will tell you nothing but if you get a double sample at least ten days apart, then you can tell whether you have a rise in titer. I do agree that veterinarians are very proficient in diagnosing the disease, but I would not want to start a panic on a clinical diagnosis in an area where we did not have the disease.

Thank you very much. [Applause]

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PANEL ON ORTHOPEDIC SURGERY

Moderator: E. A. Churchill, D.V.M.

Panel: Jacques Jenny, D.M.V.

J. D. Wheat, D.V.M.

Charles Raker, V.M.D.

William O. Reed, D.V.M.

D. L. Proctor, D.V.M.

MODERATOR CHURCHILL: The first speaker will be Dr. Jenny whose topic will be primarily the management of fractures at the race track. Dr. Jenny!

MANAGEMENT OF FRACTURES AT THE RACE TRACK

DR. JACQUES JENNY: Gentlemen, the reason I wanted to talk about fractures at the race track was simply because I do not work at the race track. [Laughter] That gives me the proper perspective to look at the situation objectively. A considerable degree of progress in the treatment of bone and joint injuries in horses was demonstrated during last year's panel discussion on orthopedic surgery: It was reported that horses can resume a successful racing career after articular surgery. Enough clinical material had been gathered to establish indications and contradications for a surgical approach to such injuries as carpal—, accessory metacarpal—and proximal sesamoid fractures. It was further pointed out that we have the basic armamentarium to cope with fractures of the long bones in horses. There are 58 reports from different countries in the veterinary literature on successful treatment of long bone fractures in large animals, however, until a horse with a radius oratibia fracture will automatically be considered a case for a hospital rather than an abattoir a drastic change of mind of the rank and file of our profession has to occur. Almost 10 years ago the justification to destroy horses with fractures for so-called "humane reasons" was questioned in a letter to the editors of the Veterinary Record. (Vet. Rec. 63, 1951, 279). The ensuing controversy, which lasted for 18 months, illustrates the problems we face very well. Many of the "unsurmountable" obstacles mentioned by colleagues engaged in race track practice could be cleared with an ounce of zeal instead of the "it is not practical"—approach. At the risk of being accused of making recommendations from afar I should like to outline a procedure to be followed if a horse suffers a fracture on the race track:

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1. Unfortunately for the injured animal and the race track veterinarian prime consideration has to be given to the crowd of people in the grandstand. Public opinion has been nursed over a long time on the conviction that fractures in horses are irrepairable. Most people feel, therefore, that it is cruel to try and treat a fracture in a horse. With the help of the press we must convince the public that people who understand the pathology of a fracture or have suffered fractures themselves know that the horse with the dangling leg might be frightened, that he must have suffered a sharp pain when the fracture occurred but that for a period of 20 minutes to half an hour after the injury the fracture area is numb and that a properly immobilized fracture is not very painful even after sensation comes back. In fact, such common equine ailments as periodic opthalmia colic or bowed tendons are surely more painful than a properly treated fracture. If we are capable of providing prompt first aid treatment, the demand that the injured horse should be destroyed for humane reasons is not justified.

2. The administration of first aid, even in accidents involving human beings, is always a show for by-standers. The unhappy track veterinarian has a grandstand full watching his performance. He must, therefore, have a crew which has been drilled for such an eventuality is order to proceed deftly and well equipped horse-ambulance which must arrive promptly. If the patient does not get up immediately after his fall he should be kept on the ground, if necessary with a dose of succinylcholin, short acting barbituate or tranquilizer. The Veterinary Hospital, Univ. of Pa., has an ambulance with a movable, thin platform which can be slipped under an animal which then is pulled into the vehicle with a motor-driven winch. If the patient is standing the loading into an ambulance, on three legs, might not be as easy. The ambulance should have a low ramp and high enough ceiling. At the AVMA convention in 1958 we applied a first aid splint [Slide] to a thoroughbred's front leg on a closed circuit television program. The appliance is based on the principle of the Thomas-Splint which is carried in every police ambulance for human accidents. Besides protecting the injured limb from further damage, it would permit the first aid crew to coax the horse into the ambulance more effectively then it would dare if it had an unprotected dangling leg.

3. Once the injured horse is removed from the track, a thorough examination of the patient is feasible. Radiographs can be taken to confirm and pinpoint the clinical diagnosis. The appropriate course of action can then be plotted by a consultation between the attending veterinarian, the owner, insurance

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adjusters and possibly a veterinarian with special training in bone and joint surgery.

While the ultimate fate of the horse which suffered a fracture on the race track might be decided on other than medical consideration, I should like to discuss for a few moments the techniques of fracture repair which a veterinarian is at this time in a position to offer. The progress made in veterinary anethesiology has really brought the repair of fractures in large animals within practical limits. Safe, reflexless anesthesia can now be obtained in horses with Ether or Fluothane. In the past many a fine reduction has been destroyed during the period of struggling which a fracture patient went through while recovering from a barbiturate anesthesia. Muscle relaxing and tranquilizing drugs have further enhanced our chances of success. A quick review of the methods of fixation seems indicated even if I repeat some of last year's remarks made on this panel: In approaching the repair of an equine fracture one must keep in mind, that in most cases the obtaining of a solid bony union does not mean success, if the patient is not capable to resume his career as a racehorse. It would seem logical however, that with refinement of techniques, which will only come through trial and error, the functional results are bound to improve. It is imperative then, that a start is made, even if the prognosis at first might be poor. In 1951 already Shuttleworth suggested, that treatment is indicated for shaft fractures of tibia, radius, ulna, metacarpus and metatarsus. Since femoral and humeral fractures are radiologically inaccessible and stablization in his opinion quite difficult, he considered their treatment inadvisable.

Based on data collected from our perusal of the literature and from cases treated at our clinic as well as the results of an experimental investigation different methods of fixation were critically reviewed. From these studies we tried to crystallize out some indications and contraindications, keeping in mind, that they will be changed and rechanged as our experience grows. As in all fracture treatment the simplest technique capable of promising a successful outcome should be the one of choice. Conservative methods should always be preferred over operative ones, but one should not adhere to this principle so rigidly as to sacrifice the patient's usefulness as a broodmare, stud, racehorse or hunter. It does not help our efforts to get fracture-treatment in large animals generally accepted, if with overzealous adherence to conservative techniques we merely assist bony union without obtaining satisfactory function.

Plaster of Paris techniques. The addition of Resin compounds to the conventional Plaster of Paris bandages has improved the usefulness of this time tested method of immobilization enormously. Not only is the resiliency of resin plaster against cracking greatly improved, but the cast becomes almost completely

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water-proof. We follow at our Clinic mostly Boehler's technique of skin-tight application using only a smooth fitting stockinette as separating medium between skin and cast. If a padding must be used, orthopedic felt is much preferable to cotton. Frequently we split our casts before the patient is discharged and hold the two halfshells together with an additional light layer of Plaster gauze or simply with adhesive type.

Plaster of Paris alone is indicated for fractures in young foals and fissured fractures of phalanges or metacarpus and metatarsus of adult horses [Slides] Mundy successfully immobilized a proximal quarter metacarpus fracture in an eight-year-old trotting mare with Plaster of Paris from fetlock to carpus. We doubt, however, that his case would have terminated favorably if the horse could not have been kept in a sling. Ueberreiter and Carnus have both reported successfully treated fractures of the first phalanx with Plaster of Paris casts. We feel, that for complete fractures of these regions in adult large animals some form of brace should be incorporated in the cast. We have used steel-bars attached to elongated branches of the shoe. The fetlock position can be adjusted and changed, if these bars are slipped through pipes which are incorporated in the cast. [Slides] Johnson has demonstrated a walking cast using a U-shaped aluminum bar incorporated in Plaster of Paris. Kendrick incorporated ¼ inch mesh hardware cloth in between layers of Plaster of Paris, and produced excellent results in badly comminuted fractures of the first phalanx in two horses.

Thomas-type Splints. Hugh Owen Thomas's principle of traction and countertraction is often quoted in the veterinary literature but rarely fully applied. The large animal "Thomas-Splints" are no exception, they mostly serve a dual purpose of coaptation-splint and crutch. However Guard has produced excellent results with this principle in horses and cattle. Rudy & Tharp immobilized a tibia fracture in a 16-year-old Stallion with a modified Thomas Splint and additional support by a sling. As to the indications we feel that these splints are more useful for the immobilization of bovine rather than equine patients since exactness of reduction and retnetion of the fragments is less important in this species, as long as a bony union occurs. We have obtained with this splint a satisfactory result in a tibia fracture of an Angus cow in her seventh month of pregnancy but failed to get a functional result despite solid bony union, in a humerus-fracture of a 2-year-old standardbred mare.

Transfixation Techniques. Immobilization using transfixation pins through the proximal fragments or both has definitely advanced the treatment of tibia and radius fractures in large animals. Reichel uses only one pin in each fragment and achieves solid immobilization by first connecting the pins with Plaster of Paris longettes and snugfitting metalbar. This assembly is then

incorporated in a regular Plaster of Paris case. Kendrick: has given very convincing evidence that the transfixation principle can produce excellent results, if properly applied. He used two transfixing pins through the proximal fragment but depended for the immobilization of the fracture upon the union between Plaster of Paris and the steelbars connecting to the pinsite. Kirk: has shown different devices of his own design which are adaptations of the transfixation principle to a frame. From the radius and tibia fractures treated at our clinic with transfixation methods we made the following experiences: Transfixation pins of quarter inch thickness will bend. A skintight Plaster of Paris or better a flat, metal connecting bar (Reichel) applied as close as possible to the skin is necessary to prevent bending of even heavier pins. Even with a handdrill enough heat can be generated to damage the bone tissue. The less transfixation pins we had to use to stabilize the fragments, the better the result. Clipping and surgical scrubbing of the whole area over which the Plaster of Paris is to be applied and not just the pinsites is essential if extensive pinseepage is to be avoided. Dusting the leg with talcum or an antibiotic power was found to be helpful in preventing skin irritations under the case. At this time I would list the indications for the transfixation Techniques as follows: Long Oblique and comminuted fractures of radius and tibia. One transfixation pin can be used through the metacarpus or metatarsus and connected to a walking stirrup in order to transfer weightbearing in badly comminuted fractures of the first phalanx.

Internal Fixation with screws and Plates: As demonstrated last year, transfixation screws are helpful to immobilize oblique fractures in the distal third of metacarpus and metatarsus. Spellman performed an open reduction on a 5-year-old flathorse with an olecranon fracture. By driving two screws through the base of the olecranon into the radius good fixation was obtained. The horse raced six months later. Pallister & Archibald used two Sherman plates and a total of 12 screws to stabilize a comminuted, compound fracture of the metacarpus in a hunter-type mare. Additional support was provided with an Aluminum reinforced Plaster of Paris cast. Schermer obtained a perfect anatomical and functional result using two transfixation screws for a comminuted, articular fracture of the first phalanx in a horse. The patient was hospitalized only for 26 days.

Intramendulary Fixation: Carney has advised the use of Rush pins for large animal fractures. From an engineering point of view the stabilization of a transverse or short-oblique fracture in radius or tibia and especially the fractures of the Olecranon would be best effected with an intramedullary appliance. The mode of approach to all three sites would also point to the Rush pin among the different intramedullary nails. We stabilized a short oblique midshaft fracture of the radius in a 6-year-old mare,

7 months in foal. The immobilization was satisfactory but the case terminated unfavorably due to causes indirectly related to the injury. Proctor has successfully nailed a transverse proximal third tibia fracture in a thoroughbred weanling using a Hanson-Street nail. Despite our poor result, we still would continue to use Rush-nail fixation for transverse and short oblique fractures in radius and tibia in preference to the transfixation method. Jonas suggested his spring splint for the metacarpus in bovines, using one splint in each medullary chamber. Ammann and Burger have both used intramedullary fixation to stabilize bilateral ramus fractures of the mandible.

The display of the varied fixation devices just reviewed, and the case reports cited, should prove, that the veterinary profession has the armamentarium to cope with fractures of the mandible, the olecranon, the radius, Carpus, metacarpus, the phalanges, the tibia, the Os calcis, and the metatarsus. As far as humerus and femur fractures are concerned Shuttleworth's statement that good results of fixation cannot yet reasonably be expected still stands. Good radiographs of these regions in large animals have not been produced. In young animals several excellent results are recorded. Guard treated a femur fracture in a 2-day-old standard-bred colt and saw the patient grow up to become a champion pacer. We have performed open reductions with Kuentscher nailing in a humerus fracture of a six-month-old standard-bred colt and a femur fracture of a 14-month-old Holstein heifer with excellent results.

General care of large animal fracture patients: This paper should not be concluded without a few remarks on the management of equine and bovine fracture patients. Front leg fractures in horses can present a serious problem if all the weight has to be shifted for a prolonged period of time on one front limb only. If possible, the shoes should be removed and the sole should be packed with clay. Silbersiepe recommended sand or peat moss instead of straw in the boxstalls, to prevent laminitis or bed sores. A suspension sling was used in many of the reviewed case reports and one might question whether the results obtained would have been the same without the sling. However, there are many patients who object to the sling specially Thoroughbreds. With the use of tranquilizers this situation might be changed. Whenever a sling is used the patient should have round the clock attendance. We made it a practice to pull up the sling only for 10-15 minutes out of every hour. This permits the patient to rest his legs and yet eliminates the danger of bedsores. Good grooming should never be neglected during the convalescence period. Much attention is usually paid to supplementary mineral supply to fracture patients. We feel that a high protein diet is at least as or even important. Especially in cases of compound fractures or fractures which have been reduced by open approach, or which are immobilized by

transfixation techniques. Every effort should be made to permit the patient as much movement as possible while the injured part is immobilized. Nothing can stimulate the rate of repair more than an active convalescence.

Thank you. [Applause]

MODERATOR CHURCHILL: In an effort to conserve time, we will hold the questions from the floor until after all of the panelists have had an opportunity to present their papers.

The next speaker will be Dr. Wheat who will present some slides and information on some cases of his. Dr. Wheat!

DR. J. D. WHEAT: We will go ahead with the slides immediately and try to save a little time.

This first case is a horse that fell with the hind leg extended behind him and suffered evulsion fracture on the tarsal bones.

Also, at the same time, there was enough tearing and injury to the point of attachment of the ligament that we have a bone tumor arising in this animal. This is an osteochondroma. We see these at this point. The epiphyseal cartilage structures are involved. They are most frequently seen on the splint bone, the hock and at the distal epiphysis of the metacarpal bone.

This animal was severely lame. The fracture was not causing the lameness. This had more or less healed down in place but this osteochandroma was the cause of pain.

Unfortunately, in this area, the amount of damage that you do in removing those is enough to prevent these animals from becoming completely sound. Those on the splint bone and distal end of the metacarpal, if you expose the base of the tumor and remove enough of the bone at the base of them, you can do a reasably good job of taking them out completely, without recurrence. It is not malignant osteogenic sarcoma but osteochandroma.

Next a brood mare that was kicked in the radius, suffered this complete fracture. This was pinned.

Now another view, the opposite direction, on this fracture. There is quite a bit of overriding. The mare was hauled into the clinic, with no temporary first aid splint or anything, just a loose leg. It was surprising that she did not displace it even more, or damage the tissues more than she did. That is probably one of the greatest factors we have defeating us in fracture repair, and that is when a horse breaks a leg, a person puts them in the trailer and hauls them in. We have had them try to put a temporary splint on them. Sometimes they will come in with a piece of cardboard on the leg, with bandage wrapped around, or something of that sort, a very ineffective type of splint. I have yet to see one come in that has been properly treated, as far as a first aid splint.

This fracture was pinned, using ¼-inch stainless steel pins, drilling through the distal as well as the proximal portion. The pins were driven through. A plaster bandage was applied, fol-

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lowed by the Kirschner blocks. The large animal Kirschner aluminum blocks were applied, and then the immobilization bar on the side. This was taken later on and shows healing, quite a bit of calcification going on. This mare made a good recovery.

At the time this happened, the mare was nursing a foal, also had been bred back and was four months in foal. This pinning was done with her on the table. The mare did not abort following the surgery. We kept her in the stall and the foal continued to nurse. It was with her all the time because, if we tried to take the foal away, she would become excited, and we were afraid it would do more harm than good. The mare was kept in slings. Some horses will tolerate slings and some won't. Unfortunately, only about one out of ten will take the slings properly. She was a mare that was of very even temperament and was left in the slings, and the foal was allowed to nurse and stayed right with her all the time.

The pins were left in the mare approximately ten weeks and the splint was removed. The mare was left in the slings an additional ten days and then taken out. Today she is getting around on the leg very well, with no interference whatsoever.

Next is a carpal fracture, evulsion type, of the distal end of the radius. You encounter more of them that involve radial carpal bone. Unfortunately, the owner did not want surgery done on this colt. They thought by keeping him quiet this would heal back in position.

Now another view of what can happen with this type of fracture. If you happen to have your cassette angled, you can miss the point of fracture. Nothing shows here in this particular film, although there is the above fracture on the back side of this joint.

This is the animal on January 6 of this year. Still have the piece here. Beginning to get some exostosis at this point. What I think will eventually happen, is, we will get lipping over here and a continuation of this exostosis. At least that has been the experience we have had with many of these, just a continual production. This horse is back in training and already is beginning to show some soreness. He is now at the point where surgery is of questionable benefit. If the chip had been lifted out at the time it happened, I would have been able, I think, to give him quite a favorable prognosis.

Now another carpal fracture, the type that we see more frequently, another evulsion type fracture. We were able to get the animal soon after the injury occurred. A thing that is important in this type of fracture is to accurately locate the piece of bone. In a lateral medical view of this sort you cannot be certain just where the fragment is. It is a matter of careful palpation plus an anteroposterior picture which, in some cases, will show the fracture, if you are fortunate to have a good film. In that way you can get it quite accurately located, so that you can make a small vertical incision directly over the chip, with a minimum

of disturbance to the surrounding ligamentous structures, go in and remove the piece quite easily.

This is a follow-up on this particular horse. You can see this is the point of fracture. There is a little exostosis at this point. However, it did not progress beyond that. The horse went ahead and raced very successfully last season. This was taken on Nov. 5th. The horse went into training soon after the first of the year and was in quite a few races and did very well.

Here is a case that is a very interesting condition. This happens to be a Thoroughbred that had a severe lameness in both legs. Every time they would take the horse out and work him, he would get extremely sore. They would lay him up for a few days, keep him in the stall, the lameness would subside. They him without forcing him along.

When the horse came into the clinic, we actually had to go ahead and block the knees to get the horse off the van, he was so sore. They raced him once in this condition by resting him in the stall five days; and then taking him out and running him. He even finished in the money. After the race, he was so sore it was not until the next day that they could even move the animal.

As you notice, the anteroposterior view of this horse does not give us a great deal of help. However, we can assume this slab is off the third carpal bone. From experience, we find the lower compression fractures are of that type.

There is one thing to remember on these carpal fractures. We sometimes get films sent to us with a diagnosis of fracture, when actually the exposure is such that you get one bone superimposed on another, and you get a sharp line down through. Some of them make such a sharp line that a person will read a film like that and call it a carpal fracture when, really, it is a normal knee. This horse had this fracture right down through the third carpal bone.

On the opposite knee he had the same thing, both knees involved. The history of this horse, as far as we could determine, was that this was of about three months' duration. You would expect to see some bone reaction and an attempt to heal at that time. However, you will note that the fracture is deep enough so that it is in communication with the joint cavity. Synovial fluid could migrate down into this tissue, at the crack, and act as a barrier against healing. If it had been a little piece pulled over the front, by now we would have seen an attempt at healing with exostosis around that point. Being in the articular cavity, the synovia prevented that from healing.

This is the method of repair we used on this horse. First, a vertical incision through the skin, directly over the site of fracture. We did open the joint space here and were able to put a probe right down into the joint. By moving the probe, you could feel the piece move. The thing making this horse so sore was

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he would get movement of this fragment, and pain resulting from that. We put a No. 5 vitallium screw directly into the fragment in order to immobilize it.

One thing, when putting these in, you want to be ready with the screw as you withdraw the drill. Have the screw ready, or at least a small bone pin or something to slip in there to keep the identity of your opening. Otherwise you can lose it, and you have to probe around and reestablish the the screw-hole opening.

Strangely enough, with this horse, the next day after the bone screws were placed, we were able to move the animal. He never did show any more of the extreme pain that he showed prior to fixation of the fragments and holding them in place. In the last follow-up we had on the animal you can see quite a good response to healing between the fragments. What the final outcome will be on this horse, I do not know as yet. I have not had a chance to try him out. It will probably be next spring before he will go back into training.

That is about all that I have to present on this subject. [Applause]

MODERATOR CHURCHILL: The next speaker will be Dr. Raker, who will present some phases pertaining to fractures of the carpus and their repair. Dr. Raker!

DR. CHARLES RAKER: We have already had presented to us some information on carpal fractures. I plan to continue this discussion, presenting some additional aspects in this field. This is not a "rigged" program, and not having planned ahead, the various panel members were not certain of the content of the material to be presented by the other panel members.

Carpal fractures do not present a serious or difficult diagnostic problem. Therefore, I do not want to dwell upon diagnosis except to make a few comments upon radiographs, to which Dr. Wheat has already made reference.

We prefer to take at least 4 views of the carpus, which allow us to rather completely evaluate the nature and extent of the pathology of the area. I think that 3 of these views are of vital importance particularly if the case is to be evaluated for surgery. The first projection is the standing lateral-medial view. The second is a flexed lateral-medial projection. The last 2 views are designated as RAO or LAO. These letters indicate the direction that the x-ray beam is applied to the carpus. The RAO projection indicates that the x-ray beam is directed from the right anterior oblique position at approximately a 45° angle. The same designation is used irregardless of the carpus involved and always indicates the direction of the x-ray beam. One other projection which is occasionally taken is the anterior-posterior, but as a rule it is of minimal value in evaluating fractures of the carpus. The lateral, flexed, and oblique views are particularly valuable to completely evaluate and localize the tip of fracture and the bone

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or bones involved. This is important from a surgical aspect both from the standpoint of invasion of the joint in the proper area and determining whether surgery is indicated or contra indicated.

[SLIDE] In diagnosing carpitis and fractures of the carpus and in performing surgery upon this joint, one must be familiar with the anatomical structure of this joint. Perhaps a quick review as depicted on this slide might be in order.

As you look at the front of the carpus, you see designated the medial and lateral surfaces. You will notice that the joint space between the radius and the proximal row of carpal bones and the joint between the proximal and distal rows of the carpal bones are the only two in which flexion of the carpus occurs. You will also note the presence of transverse ligaments between the bones comprising the proximal row of carpal bones. Similar ligaments are present in the distal row of carpal bones, and in addition, longitudinal ligaments bridge over the joint space of the carpal-metacarpal joint. These ligaments, of course, prevent opening or flexion of this joint. Also, it is important to note that the joint capsule between the radius and first row of carpal bones is separate from the joint capsules bridging over the first and second row of carpal bones and the carpal and metacarpal bones. Frequently the joint space between the latter two joints communicates.

Because of these ligamentous and capsular attachments, much of the pathology detected on radiographs is evidence of damage to these structures, and as healing takes place, new bone is deposited. Usually these new bone deposits are removed from the margins of the joint space, and thus do not interfere with function.

In our experience, most of the chip fractures involve the radial or third carpal bone and less frequently involve the intermediate and fourth carpal bone. The chip fractures of the distal end of the radius are also relatively common. We use the term "slab" fracture of the carpal bone to designate a fracture extending completely through the body of the bone from one joint space to an opposing joint space. In comparison, we use the term "chip" fracture to designate a fracture wherein a small segment of the bone is involved, usually along the margin of the joint space.

It is also important to recognize the location of the tendons and tendon sheaths. Should a tendon sheath be opened accidentally during surgery on the carpus, it should be sutured with a #00 chromic catgut with a swedged-on atraulic needle. Failure to suture an exposed tendon sheath may result in the loss of flexion of the joint. Fortunately, most of the pathology of the carpus involves the dorso-medial surface of the joint, which is relatively free of any important tendinous structures.

Directly on the front of the joint, we note the extensor carpi radialis with the common digital extensor lying lateral to this tendon and, still further to the lateral side, the lateral digital

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extensor tendon. Therefore, if you must approach the carpal joint from the dorso-lateral surface, it becomes more difficult and greater care must be taken to avoid opening the tendon sheaths.

[SLIDE] This slide of fore leg of horse in full flight is shown as one example of why horses break down. In my opinion, the main factor is one of trauma, and carpitis is usually the result of traumatic injury particularly during the late stages of a race when muscle fatigue is present. In examining this slide, you can readily observe the over extension of the carpal joint, which greatly increases the stress placed upon the anterior margin of the joint. Note the position of the fetlock joint which has dropped almost parallel to the ground and, in fact, appears to be in contact with the ground. There are many contributory factors such as fatigue, age, hard race tracks, mud, improper condition, and so on. I do not plan to discuss these factors at this time.

Traumatic injuries to the carpus may involve damage to the capsules, ligaments, or tendons and bone. Exostosis or osteophytes in areas removed from the margins of the joint space are usually an indication of damage to the soft tissues. During the healing process, new bone is deposited and is shown on a radiograph as a new bone growth. By the time the repair of this type of damage is completed, as a rule, it causes the horse no disturbance and produces no particular defect in gait as long as it is removed from the margins of the joint space.

Dr. Grice, Hospital of the University of Pennsylvania, Orthopedic Surgery, states that evidence of this type of lesion in man is a favorable sign because it indicates healing. However, once degenerative disease involves the margins of the joint space or degeneration within the joint space develops, which involves the articular cartilages, we then have a condition which will disturb function and the usefulness of the horse.

It is necessary to have good radiographs to determine the presence of destruction of the articular cartilages. Infection of the joint resulting in a septic arthritis, regardless of how induced, promotes a leukocytic response. The leukocytes produce an engyme which can lyse or dissolve the cartilage. It is a known fact that articular cartilages heal slowly and frequently inadequately. Thus, if we have evidence of arthritic changes within the joint, the condition is usually incurable from the standpoint of racing soundness, and surgery is frequently contra indicated.

This slide was sent to me by one of the members of our association. It was sent to me in July. The original pictures were taken in May, at which time we see evidence of a small chip fracture of the radial carpal bone. This case would have been a good surgical risk, but instead of resorting to surgery, the carpal joint was injected with cortisosteroids and the horse was raced. It soon became evident that the steroids no longer produced the desired relief of the signs of lameness. Thus, additional radio-

graphs were taken in July. We now note extensive degenerative arthritis of the carpus with involvement of the joint space and extensive proliferation of new bone. At this time, the case is a poor surgical risk and surgery is contra indicated. The steroids act as powerful anti-inflammatory agents and by removing the pain and restoring function, we allow the horse with a fracture, such as shown here, to induce further damage to the joint often to the point that it cannot be repaired. Whether the degenerative arthritis shown is purely the result of continued trauma in the presence of a fracture or associated with infection in the joint, I am unable to determine. I would seriously advise that the use of steroids in a joint should be preceeded by a careful clinical and radiographic study of the joint to determine the nature and extent of the pathology, and then decide the proper course of therapy.

This is a radiograph taken in September on the same horse. It is now evident that there is a complete involvement of the joint space, and there is no flexion of the carpus in this area due to the fact that the front of the joint space has been bridged by new bone deposits.

There are other therapeutic measures besides surgery and steroids such as ultrasonic therapy, x-ray therapy, blistering, firing, and so on, with which we are all familiar.

I will confine the remainder of my remarks to the surgical management of fractures of the carpus.

These pictures were taken of a cadaver. We did not use the plastic Vi-Drape Dr. Jenny mentioned, but used dish cloths to cover the skin fastening them to the margins of the skin incision. The area is clipped, scrubbed, and adequately prepared for aseptic surgery.

The skin incision is curved with the convexity to the medial or lateral surface of the joint depending on the area invaded. The convexity should be as close to the side of the joint as possible.

We then reflect the curved flap of skin, which exposes a larger area for entry into the joint. The incision into the capsule does not lie directly under the skin incision, a greater area is exposed, and less tension will be applied to a curved suture line than to a straight one. This is particularly true when incising over a movable area such as a joint.

After the skin edges have been draped, a small incision is made into the joint as close to the fracture as possible as depicted on the radiograph.

Some of the special instruments used include bone currettes, bone elevators(large, small, and blunt), retractors, chisels, and rongeur bone forceps.

A Bishop orthopedic drill and saw combination, which both oscillates and rotates, is an extremely valuable piece of equipment. We use it primarily for drilling holes for pins and screws

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in fracture repair work. It can be autoclaved and is completely portable.

This radiograph shows a chip fracture of the distal end of the radius and a few small fragments from the proximal row of carpal bones with some degenerative changes of the margins of the joint space. We have operated upon several cases of this type, and it is not difficult to remove the chip and clean the margins of the joint space, providing an aseptic approach is used and maintained throughout the surgery.

This radiograph shows a small fracture of the radial carpal bone without evidence of degenerative joint disease. Surgery is indicated and the prognosis is favorable.

This radiograph was taken while the horse was still on the operating table. We frequently employ this procedure to check our results before closure of the joint.

I owe thanks to Dr. Teigland for these radiographs. This horse had an old chip fracture of the distal margin of the radial carpal bone with a few degenerative changes in the region of the fracture. The chip was removed and the margins of the joint space debrided. The horse made a satisfactory recovery and is now racing in a higher class than before surgery. You will note that the progressive radiographs taken in 6 months show no recurrence of the degenerative lesions on the margins of the joint space.

This radiograph shows a clean, relatively small chip fracture of the proximal margin of the third carpal bone. This was an excellent surgical case and was operated upon, and the horse has raced successfully.

This is a postoperative view of the previous case. You will note that the small chip of bone has been removed and the joint appears to be clean.

This radiograph shows a "slab" fracture of the third carpal bone. The size of this type of fracture is variable. With the carpus fully extended, the fragment often moves proximal so as to lip over the front of the joint space.

This radiograph shows an oblique view of the above case and more clearly depicts the size of the fracture involved and, in addition, definitely indicates the bone involved.

The next radiograph is again of the above case and depicts the carpus in the flexed position. You will note a complete reduction of the fracture with the carpus in this position. This was actually the first case we had seen. We have now repaired over 15 similar cases by the insertion of a vitallium screw. The hole is drilled and a guide pin is inserted. A radiograph is then taken to determine satisfactory placement of the hole. Then the screw is inserted keeping the carpus in the flexed position.

This radiograph illustrates the screw in place and you will

note satisfactory reduction. Healing was complete at approximately 4 months, and the horse raced well for 2 seasons.

I do not have available complete statistics on these cases, but of the first 8 repaired in this manner, I know that 7 are racing sound to the satisfaction of the owner and trainer.

At the present, instead of opening the joint in an uncomplicated case, we make a small incision over the third carpal bone, drill our hole, and insert the screw with the carpus in the flexed position. This can only be done in fresh cases, providing satisfactory reduction is evident on the radiograph taken with the carpus flexed. If complications are encountered, the joint space is opened and a more complete investigation is conducted. These horses, as a rule, will be walking nearly sound the day following surgery. If it becomes necessary to open the joint or in the presence of extensive degenerative changes, one must naturally expect increased signs of lameness following the surgery.

I have not had the time or opportunity to gather all of the statistics together on the cases of carpal fractures on which surgery has been performed. It is extremely difficult to get satisfactory follow-up reports. We have operated upon more than 100 carpal fractures of various types, and I will give you a rather brief run-down on the approximate result on the first 40 cases.

In cases with uncomplicated fresh chip fractures, you can expect 75% of the cases. Uncomplicated fresh "slab" fractures should respond in 80% of the cases surgically managed. Complicated or old "slab" fractures respond in approximately 50% of the cases.

Degenerative carpitis with the degenerative lesions along the margins of the joint space respond in approximately 50% of the cases if carefully selected. In general, this type of pathology is not a good surgical risk and there is evidence of recurrence of the pathology in many cases.

MODERATOR CHURCHILL: The next paper will be by Dr. William Reed who will speak on correlation between clinical and x-ray diagnosis.

DR. WILLIAM O. REED: As you all know, x-ray is almost a must in diagnostic procedures for orthopedic surgery. However, it is our feeling that many times we overlook the physical characteristics of the animal in motion, and sometimes we get a little lax about that. In other words, we might be thinking a horse is lame in the foot; x-ray the foot and find that he is lame somewhere else.

We will follow the movie with some x-rays to show you characteristic types of lesions that we find in the foot and on up.

[MOTION PICTURE] This is the start from the gate, in slow motion. You can see the stress is unusual at this particular time.

This is a horse in motion. This happened to be Nashua when

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he was the leading money winner of the world. Here he is in motion on the track. He had excellent action, as you can see. I pilfered the film from Hialeah. It does not show everything we especially like to see, but it is the only thing we could get, under the circumstances.

Here is a characteristic thing we call, for lack of a better term, ringbone. We see these things and immediately think this is typical ringbone. X-ray shows later on it is not typical ringbone. It does not have exostosis but, for lack of a better term, we have always called them that. They are usually characterized by high heel and eventually suspensory involvement. They do cause a lot of soreness. From the exostosis standpoint, there is usually none there. This is an A.P. of it, and it shows distinctly there. Dr. Jenny does not agree that this should be termed subluxation.

Here is a typical ankle lameness. This horse is lame in the right leg, as you can see.

We usually start by seeing them move like this, and then flexing the ankle like this. You see how lame he is there.

Here is typical osselet. Here is an ankle condition you are all familiar with. You see enlargement of the ankle, hot, painful, sore. You flex them; they don't want any part of it. If not treated, they get progressively worse, end up with an ankylosed joint, are extremely sensitive and deteriorate in value. This horse was operated on for bowed tendon. He won, roughly, a quarter of a million dollars, not after he was operated, but he keeps knocking around, and gets a pot. He is running for about $7,500. He is relatively sound. In other words, you have to bear in mind that he is serviceably sound, and able to go ahead.

Here is a condition of the knee. You notice how apparently knock-kneed he is. They are congenital knees. You never do anything to improve them. They get only one way, and that is worse. I would imagine that sometime early in developmental life there was some predisposing cause for it, but by the time we see them there is very little that can be done.

Here is what appears to be typical foot lameness. You note how sore this horse is when he puts his foot down. You would swear he was lame in his foot. You could not be convinced it was anything else; in fact, I wasn't.

The first thing you would do, as a rule, would be to go ahead and x-ray that, and you would be dead wrong, as you will see later on. The typical characteristic of raising the head when the foot hits the ground, very little abduction or adduction of the leg here, pretty much a straightforward position, but very lame.

You notice how easy he is on the knee. He flexes the knee, does not particularly care to bear weight. In some foot lamenesses you think they will stand on the toes; some of them show pointing action, different types of characteristics.

This was one that could easily fool you. You would swear,

from the original pictures, it was a foot lameness. However, by use of pressure test here, first gently, to try to pick up any pinpoint foci of soreness of sensitivity, it does not show any soreness. Then by hard pressure, still no soreness or sensitivity.

Now watch this, watch what happens to the horse. Repeated the same procedure; the same situation. Flexion of the ankle, no soreness. Probably wasted a set of x-rays on that.

If you go back to the radiograph and check, you can see how sore he was. This is a little dark, but this is a typical knee type of lameness. You notice the abduction of the affected leg, which is the left leg. He is toeing out considerably, wants to bear no more weight than he normally can; wants to get off of it as much as he possibly can.

We will se an x-ray pretty soon of this horse. He had a huge fracture which was removed, and the horse is sound now.

We run into the old question of sore horses. Here is a two-year-old, one of the leading two-year-olds in the country this year. He has been sore all summer long, ankles, feet, legs, all over, completely cold, no sign of anything. He does not look too bad here.

We blocked the feet, and he was just as bad as ever. We see an awful lot of these sore two-year-olds. Why, I don't know.

Here is a typical wobbler. A lot of people have not seen wobblers. They frequently ask what they look like, how do they go? I never can quite describe them. The best thing I can say is that this is the way they look, as someone says, they are all out of time. It is noticeable when you pull them up; they get all incoordinated. When you turn them, they walk wide behind, show no strength in the tail. The tail seems to flop along behind them. Yet some of them, believe it or not, get along pretty well, and I know horses that have raced afterwards. They do not race too long.

Usually the first history you get is the fellow comes to you and says, "This horse acts awfully awkward." You question him and he insists he has been awkward all of his life. Usually they are not, though.

Backing is a problem. That is always a problem. Here is another one. Some of them, when you walk them, they do not walk too badly. You think they are big, old, awkward colts, undeveloped, and not typical wobblers. The more you walk them and the farther you go with them, the more they show it. I like to spend plenty of time with these because I think snap judgment on them is wrong. It has been that way for me, anyway.

Notice how wide his hocks are. Turn him again, it is not easy for him. Watch him when he trots. He acts like a drunken man.

Here is a rather interesting thing. This is not in the line of orthopedic surgery, but this was a foreign body thing that some of you know about. This little fellow is pointing to a scar where I made an incision to remove a foreign body. Actually I did an

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esophagotomy on this horse and will show you the x-ray pretty soon of what we took out. I hope it never happens to any of you. It happened to a colleague of mine, and it has been the source of a little bit of embarrassment. That is the extent of the incision.

Here is what we consider to be a horse excellent conformation. This is Sword Dancer, the horse of the year this year; wonderful disposition; has feet and legs like iron. Conformation-wise, he is very hard to follow. Stands very true on his legs, is a very deep-hearted, excellent quarter horse. Probably if they were all like this, our problems would not be nearly so great.

Here he is in motion, going away from you. You notice the hind leg motion or action; it is about as good as you need. Coming to you, you notice how straight he moves. That is completely natural for him.

Here he is pulled up in a standstill. He does not toe out, toe in or show any unusual stress at any particular place. I think a lot of times these conditions are a result of stress due to architectural deformities. Front legs particularly good, good shoulder and excellent hindquarter on him. This was just recently, actually about ten days ago, or a week ago.

This is a completely new trick to me. Here is the pseudo-ringbone we saw a while ago. You notice the apparent subluxation of the joint. What causes that I do not know, but I do feel that most cases are associated with the high heel in a horse, and are associated sooner or later with suspensory trouble.

Coming on up the ankle, we see various types of fractures, chip fractures. This is a chip fracture of the sesamoid bone.

Here is a little bit bigger fracture. It is my experience that this kind does not cause quite as much trouble as the little chip ones that work in the joint and are painful. This one was surgically removed, and the horse won two races this year.

Here is a shattered type. Both sesamoids are completely fractured. There is no chance for those surgically or otherwise, in my opinion. If you want to save them for stud, you can put a cast on them and leave them alone, and they will hobble around, but will always be semi-crippled and end up with an old ankylosed ankle, too.

There is a typical proximal fragment. These are relatively easy. They are not too difficult.

This sort of breaks your heart. Here is what we consider typical osselet on x-ray. You can begin to see the osselet. You can see the fuzzy outline in here, and there are prominent exostoses starting.

Here is what we are trying to show you, and that is sesamoiditis on the back part of the sesamoid. They are sore, too. It is proliferative periostitis and ends up with exostoses around, and eventually get sore.

Here is typical fracture of the cannon bone right down into

the joint, and they are tough, particularly if they are a complete fracture like this. We have tried some screws. I think maybe there is one in here of a screw. Here is one where we put three screws in. That came out pretty well, too, believe it or not.

Typical splint bone fracture. You have all seen those; you know how they are. They vary in location, of course.

This is a postoperative one. Here is the distal portion. This was, I would say, roughly, a month and a half later. These are the ones that will cause you a lot of trouble. You look up one day and the horse has a great big, edematous type of leg. They are edematous; that is the best way to describe them. As you can see, the splint bone is separated from the main metacarpal bone. Dr. Jenny tells me he thinks in these cases there are fractured lines. I am not so sure but what he is probably right, but he feels like you are justified in removing them. We have removed several of them, and they are perfectly all right afterwards.

Going on up to the radial carpal bone, here is a chip fracture off the radial carpal bone. They are simple, easy to remove, if you can locate them as Dr. Wheat and Dr. Raker mentioned a minute ago.

In the flexed knee, this is osteochondritis dissecans or free body in the joint itself. Originally, these fractures are pieces of avulsed bone that have broken loose completely and are lying free in the joint.

Here is what I would consider the first stage to that. Then the next stage is they are free in the joint. The horse will race pretty well and, all of a sudden, come back lame. Sometimes they can't begin to cool him out.

This looks like it is partially attached to the bone. This is a slightly different angle. You can see the freedom there. That was operated on all right.

Going to the hindleg, this is a little unusual. We have two views. The next one might be a little easier. The second phalanx has a little chip fracture up in here, over the circled area. We then underexposed it. You can see how much easier it is to pick up. Sometimes I feel, if you are not positive, if you change your exposure, I think more frequently to under-exposure rather than over-exposure, you can pick up many of these things a lot easier.

That appeared to be rather innocent. Here it is and it is a pretty good sized piece. From the side view you would think that all you had to do was to make a little incision. It was no easy task to get out. The mare is sound now and is all right.

Here is a fracture off of the lateral malleolus of the tibia. The history of this was that the horse came out of the gate, hit the side of the gate and, of course, was lame. On palpation, you could literally move that bone around like a pair of dice. It was very easily palpated just beneath the skin. Fortunately, it was very simple to remove. We simply made an incision in the skin, picked

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it up, and once we freed it of its attachment, the thing just popped out.

Getting back to the controversial fibular thing, here is one, fortunately, that we were able to do an autopsy on. It did have a fractured fibula. So, in my mind, certainly, this was a fractured fibula.

I showed the filly a while ago with the foreign body, on which we did an esophagotomy. Here is what it was. How would you like to have that happen to you?

This is the first cervical vertebra and the second cervical vertebra. In this darkened area is the trachea, and that is the end of the balling gun. We tried to get that out, put the filly to sleep, and take it manually. I couldn't begin to do it. We ended up doing an esophagotomy. I hope that doesn't happen to any of you. That is all. [Applause]

MODERATOR CHURCHILL: We have one more speaker on the panel. The next speaker will be Dr. Proctor who will comment on a movie portraying sesamoid fracture repair and also show some slides.

DR. D. L. PROCTOR: As low man on the totem pole today I have strictly had all of my big guns stolen from me. I think these gentlemen have covered the question of fractures very thoroughly. I am afraid I just have a little more of the same.

I have listened as carefully as I could to their dissertations. I do not want to take active exception and certanly do not want to set myself up as a gospel soothsayer, but there are certain things that I do differently. In this free world there is a legitimate difference of opinion. My approach is strictly that of a practitioner who does equine surgery, and I try to do things in as economical and efficient, easy manner as I can, without by-passing the fundamental principles of asepsis, which I learned the hard way can't stand to be by-passed.

I notice a great number of slab fractures have been shown. I was initiated to the science of slab fracture removal by Dr. Jenny and Dr. Reed. Being a hard-headed kind of Irishman, I watched the way they did it and I liked it, and I have not seen any reason to quit. I still take out the pieces. I feel that we do not get as much calcium deposition as they seem to feel they get. With the piece completely out of there, I have had almost as good results; not on as many cases, but I have got three good winners now that have had slab fractures completely removed. Slab fractures, too, are a little difficult to get out, because the extensor carpi radialis lays right over the central carpal bone, and it is quite difficult to free it. I have to use a periosteal elevator. On occasion we have even broken the sequester in two, trying to remove it.

Another thing, there has been a good bit of emphasis put on the size of the incision, and not interfering with the joint capsule, and loss of synovia. I am kind of a reckless man when I

get in there. I don't like to work in small areas. I never bought half a pint of whiskey in my life. [Laughter] I open the area up. I feel skin incisions heal side to side and not end to end, and a 12-inch incision will heal just as quickly as a 6-inch. I cannot understand the rationale of Dr. Raker's explanation for making a semicircular cut in the carpus. I personally feel that a straight up-and-down incision is less likely to interfere with nerve and blood supply. As I said before, I make a liberal incision, and I can tell you from my own experience that the loss of synovia, in my opinion, is not significant. Empty the joint if you like, nature will put it back.

[Motion picture] This animal is in our presurgical stall. The leg has been done up with bichloride of mercury, 1:500 for 48 hours. We try to do our clipping and shaving prior to the operation.

The animal is taken into this wash stall. His feet are picked out, and the antiseptic pack is removed. I do not have a trained anesthetist. I am not sure I would know how to train him, if I had one. I have learned the hard way, that the less general anesthesia I use, the fewer fatalities we have.

We have adopted the practice of local anesthesia and sedation as a suitable means of doing this type of joint surgery.

Here I have blocked the volar distribution. The animal is being placed on the table. We have the odd one that gives us a hard time but the majority of them go on without any difficulty.

I do not hesitate to open that skin and get a good view of where I am going and what I am trying to do. I am a great believer in multilayer openings and multilayer closures. I think one of the keynotes to good healing is a multilayer closure that obliterates all air space.

I was intrigued with Dr. Reed's plastic drape and was much in favor of it, but they cost $70, anl I figured I could wash a lot of cloths for that. I am too lazy to sharpen knives, so I use a detachable blade. [Laughter]

I have made the incision into the joint capsule. You can see the synovia escaping. As you look in there, it is at once apparent that we do have definite joint damage. The interior of the joint capsule is sort of a brilliant orange color rather than the nice, healthy pink that you associate with normal joint surfaces. You can see the difference in color right at the end of the forceps. The percentage of animals with apical sesamoid fractures surgically removed that go back into training, and race successfully, is very high, about 60 to 70 per cent. There you see the removal of the apical fracture. We curet, try to clean up the area, and make sure there are no spicules of calcium or rough areas. I use stainless steel wire, and interrupted sutures, and try to make as firm a closure of the ligaments on the side of the joint as I can.

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I make no particular effort to close the joint capsule. I simply close the ligaments over the top of it. I do it very firmly.

The main point of technic here is to cut off the pieces of stainless steel sutures just as close to the knot as you possibly can. If there should be a sixteenth or thirty-second inch of free steel, I take my needle forceps and try to smooth them down, so there are no sharp points protruding.

I do not use any antibiotic locally. We do put them on penicillin and streptomycin systemically for about ten days postsurgically.

We have closed the annular ligament. For the fibrous tissue on the lateral side of the joint capsule, we use 00 gut and make a fascial closure with the reflected fascia.

I use the remainder of the gut that I have left over from the fasical closure to approximate the edges of the skin about every inch, with interrupted gut, I close the skin with continuous silk. Unless you do approximate the edges, you are going to end up with a little skin left over on one side or the other of the incision occasionally.

The leg is covered over with sterile 4 x 4's, and we put an Ace bandage on. The animal is put back on his feet. It makes a wonderful impression on your client to see his horse on his feet and walking away.

You will notice the round rollers on each side of door opening. These have saved many a hip contusion.

We are back to the radiograph technic.

This is an injury on the anterior surface of the cannon bone and is characterized by a tremendous amount of calcium deposition and enlargement. I feel this is a subperiosteal hematoma. Had I gotten it early, it could have been aspirated. As it was, we had to make an incision and curet; healing has been fair.

This is a horse called Royal Vale. He had a chipped fracture at the distal end of the radius and was one of the most satisfactory cases that we worked on, inasmuch as we literally popped it out like a blackhead. I had a call from California the day before yesterday. I feel sure he is going to be race horse material. Royal Vale won on Feb. 20, 1960.

Gentlemen, thank you very much. [Applause]

MODERATOR CHURCHILL: Thank you, gentlemen, for your fine presentations.

Are there any questions from the floor regarding these presentations?

DR. O. R. ADAMS [Fort Collins, Colo.]: I have two questions. One is I would like to know the type of tourniquet Dr. Proctor used. Hemostasis was excellent.

Do you put plaster cast, following surgery, on the carpus to restrain the joint?

DR. PROCTOR: The type of tourniquet is simply a piece of rubber inner tube, that I cut round and round, like you used

to do when you were taking leather work as a Boy Scout. I do believe it is important to drive the blood out of the leg. That is the fundamental precept in putting on tourniquets. I use a 4-inch bandage at pressure points. Then we release our tourniquet and attempt to tie off the big vessel. By and large, this gives us a good, clear field to work in, and we are able to see exactly what the extent of the pathology is, and what we are doing.

What was the rest of the question?

DR. ADAMS: Do you put plaster casts on, say, the carpus following removal of the chip?

DR. PROCTOR: I do. I feel that, to protect the horse from himself, it is necessary to immobilize that joint. I use orthopedic felt, and cover the accessory carpal bone, and the points on either side of the radius where there is nothing but skin and bone, and we put a plaster cast on. However, we leave them on only about ten days. At the end of that time, the skin healing is complete. If you leave them on any longer than that, you get an exorbitant number of pressure necrosis spots.

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DISCUSSION OF INSURANCE PROBLEMS

Moderator: Ed Churchill, V.M.D.

Panel: Mr. Warren Wright

Mr. David Denker

To start this off, I would like to call on Dr. Jacques Jenny to give his views on the discrepancies that the veterinary profession now feel exist in our relationship with the insurance companies.

Gentlemen, Mr. Alex Bower of Lexington published in the Blood Horse a reply to my remarks on this panel last year because he felt that I wrongly accused the Insurance Companies of inhibiting progress in fracture treatment in horses. I am afraid I must have expressed my feelings on the subject very poorly. I simply tried to make the point that if we are to make progress in the treatment of fractures in horses we should be able to gain experience on the large number of "cheap" horses which are destroyed without attempting a repair instead of being called only to repair fractures in very valuable horses. Techniques used in fracture fixation in small animals and in man cannot simply be adapted to the larger species. While the principles are the same, the methods of application have to be developed by trial and error until fracture treatment in horses becomes standardized and predictable. The large number of "cases", man himself being the "guinea pig", has made the physician so competent in treating human fractures. Likewise the small animal veterinarian has an opportunity to treat large numbers of fractures and has developed an equal proficiency. The equine practitioner is in a particularly difficult position. For him economic rather than medical considerations dictate the treatment. If the injured animal happens to be a gelding or a colt with no stud-value and little chance for a comeback even if the broken bone heals, it is understandable that the owner has no interest in treatment, especially if he can collect an insurance sum if the horse is destroyed for human reasons. Thus the insurance companies become owners of horses which have no value except for promoting the advancement of fracture treatment. I would propose that Insurance Underwriters make such horses available for treatment rather than just have them destroyed. The patients would receive proper first aid. Thence they would fall into four categories:

1. Necessary operations would be performed and if the success would be doubtful, the patient would be destroyed without regaining consciousness.
2. If the patient has a reasonable chance and can be saved but

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does not become really sound, he will be destroyed after proper evaluation.

3. Horses which are saved and sound but not capable of pursuing a successful racing career are disposed of for their best possible use.
4. Horses capable of again pursuing a successful racing career are resold for this purpose.

An agency is to be formed which will underwrite the cost of treatment. Funds for such an agency are to be secured privately and any possible revenue from the disposal of horses would be channeled to this agency. A committee should be appointed by the Association of Equine Practitioners to correlate efforts made in different parts of the country and to make recommendations as to the most promising procedures to be followed.

Gentlemen, that I submit this as a rough outline, and I would be eager to hear your comments. Thank you. [Applause]

MODERATOR CHURCHILL:

Now we are fortunate in having two well known representatives of major horse insurance agencies in this country with us. We would like to have their comments.

First, Mr. Wright.

MR. WARREN WRIGHT: I honestly feel that I should be an x-ray plate. then you could understand me better. However, as a representative of the underwriters of Lloyd's of London, I think the idea proposed by Dr. Jenny is the solution to a problem that faces all of us. How well it will be received by the underwriters, I cannot tell you.

The problem with the underwriters, and I do not know how many of you know it, is that they have been losing money due to fractures and the resultant destruction at race tracks. They lost, although I cannot tell you the exact figures, I think in excess of $40 million dollars on marine business, which includes horses, in America in 1958. Our American companies have an investment portfolio, and the profits are better than the losses.

As you know, the underwriters are simply a group of individuals and syndicates, and they put up their personal fortunes. So they have no investment, you might say, to recoup the losses from the livestock mortality.

Now our basic problem, as I see it, and I believe you will agree with me, is the policy as written today is a death policy. It provides for destruction by humane means, or natural or accidental death. There is no give or take in the policy. That has been our biggest problem. Our underwriters have come over here to see us, and we have gone over to London to see them. This problem has been discussed. I will admit there has been no concrete proposal such as Dr. Jenny put forth now.

I want it clearly understood that I speak only for myself,

but I think, perhaps, our industry can work with your committee that may be set up to go into it, in some way.

The problem of reimbursing the owner for his horse, should the committee take it over; the problem of Lloyd's agreeing to all of his, is a large one. I do not think it will come immediately but I hope, for the sake of all, we will find an answer to the problem.

If you have any further questions, I believe it will be best to answer them directly, instead of trying to go into generalities.

MODERATOR CHURCHILL: Mr. Denker, would you care to make any comment?

MR. DAVID DENKER: I think Mr. Wright has aptly expressed the situation with Lloyd's of London. It is unfortunate they are not an individual company. They are a group that has banded together, and it is difficult to get them all together to decide on one set procedure. There are various underwriters, comparable to 100 American companies. To get them all to agree to one set procedure is a difficult thing.

In the United States, there is one company now, the Animal Insurance Company, that is attempting to effect the possible salvage situation, but there are also problems such as when does the university take over the animal, who is responsible for the shipment from the farm?

I think they would welcome any suggestion such as this, to answer their problem. This problem is probably bigger to them than it is to the veterinarian. They are losing money; you boys are losing the animals on which to practice. I think that is the whole thing in a nutshell.

DR. J. E. BURCH [Miami Shores, Fla.]: There is one question I would like to ask either one or both of you gentlemen, and that is this: Has there been any provision with Lloyd's of London set up where we can have continued coverage on these horses for such period of time as you can determine whether they will successfully recover from this type of surgery?

MR. WRIGHT: Not to my knowledge.

DR. BURCH: Has there been any attempt to set that up?

MR. DENKER: It depends on the nature of the condition. In other words, is it due to accident, illness or what?

DR. BURCH: Fracture.

MR. DENKER: Veterinarians do not agree. One says destroy; one says don't destroy; one says operate. We feel, basically that, if it is necessary to save the life of the animal, in other words the horse would be put down if we don't operate, operation will be consented to, and there is coverage, if it is necessary to save the life of the animal.

DR. BURCH: You missed my point. Many times it is not a matter of life and death; it is a matter of recovery for soundness or not soundness.

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MR. DENKER: If it is an operation not necessary under a policy, the policy will automatically cease, if the operation is attempted.

MR. WRIGHT: Unless the underwriters are notified prior and given full details of the operation. The trouble is in a fracture case it takes so long. You are running from two days to eight days on such a problem.

MR. DENKER: In other words, the policy will not cover an operation which is not absolutely necessary for the life of the animal. We do not insure racing performance. It is not an accident and health policy. It is strictly a mortality policy. The rates are promulgated on that and, as it is, they are losing money.

DR. BURCH: It is obvious to you gentlemen, from watching these pictures, that many times on the sesamoid fractures and carpal bone fractures, they are not a matter of life and death but there is an immediate relief, many times, of the pain and symptoms and inhumanity that goes with that following an operation. We are put in the embarrassing position of advising clients who have their money invested, what to do. There are many of us that are on the fence right now.

MR. DENKER: So are we. We have to go by the policy terms and conditions, unfortunately.

PANEL DISCUSSION OF PROBLEMS IN THE STANDARD-BRED

JAMES T. O'CONNOR, V.M.D.

JOHN R. STEELE, D.V.M.

BERNARD F. BRENNAN, V.M.D.

Since the advent of all-weather race tracks, racing from March to December, 2:05 miles in two-year-old pacers and trotters, it has become a great challenge to keep the standardbred race horse sound, with particular attention to the rear extremity. The drive or push derived from the hind leg of the trotter or pacer has caused many sites of primary trouble. It is these that I have been asked to discuss.

Let me impress everyone that many times trouble starts or develops in a hind leg resulting from a front leg lesion. As a matter of fact, in most cases it is necessary to rule out the front end before a positive diagnosis is made and treatment is started. The very fact I do not think can be stressed enough.

In general, most horses show characteristic symptoms when they are hurting in the hind leg. There is a shortening of stride, a tendency to travel on toe; a tendency to travel away from the affected part. These characteristics cause a hopping or hiking gait behind. This is most accentuated in the trotter, due to the extreme action when at fast gait. On the pace, horses can be gaited that have limited action, whereas on the trot, the hiking is very pronounced.

In discussing rear leg lameness, I am going to break it up into four parts: hip area, stifle area, hock area, and ankle-foot. If it could be as clear cut as this, it would be easier to discuss, diagnose and treat. However, in most cases it is a complex situation involving more than one of the areas, especially after a lameness or soreness has persisted for sometime.

In establishing a diagnosis in a standard bred, it is necessary to use all aids: x-rays, nerve blocking, common sense of mechanics, and plenty of time and observation. It is necessary to examine the horse at rest and on the track. Careful palpation of the extremity for heat, swelling and soreness is a must.

In low soreness, that is foot, pastern and ankle, the animal travels with a decided shortening of gait. The flight of the foot may be straight, or there may be bearing away from the affected side. At rest, the animal usually will rest and not bear weight on the affected member. At work, the horse most often becomes worse with exercise, depending a lot on the lesion.

A horse with lameness low usually will show a considerable

limp, with a dropping of the opposite hip, much as you would get with a front leg lameness. You get the dropping of the hip in reference to dropping of the head.

Never block at the sesamoid level helps to localize it to the lower extremity. Holding the ankle in a flexed position, without flexing the hock, and then starting the horse off suddenly, with the horse going extremely lame, is a very definite or positive test for sesamoid soreness and ankle soreness. However, it has been my experience that, when you get a positive test such as this, do not always think that you have just trouble in the ankle. With foot soreness and pastern soreness, you get the same positive type test.

With hock lameness, it is characterized by the animal resting the affected leg on the toe. When starting out, the animal walks on the toe but gradually starts hitting flat. Wearing out of the shoe on the toe is quite a characteristic symptom of hock lameness. It is something that I feel, in time, if you have seen enough of these and have blocked these and know it is in the hock, you can pick them up by just watching a horse go on a race track. Carrying the body away from the affected area is common. Thus the bearing in and out, when cornering, or on straightaways. Flexing of the hock, and starting off suddenly, with indication of lameness, is a positive sympton.

In diagnosis of lameness in a hock, I think there are two things that we run into commonly. One is inflammation in the cuneon bursa and the other is a strictly joint lameness. If I am convinced that I am dealing with a hock lameness, I usually go about it like this: I like to see the horse move, to demonstrate the lameness. I like, first, to block the cuneon bursa with Xylocaine. Let this have a chance to take effect and about ten or fifteen minutes later take him out on the race track and see whether you have any improvement or not. If you have improvement, I think definitely the surgical approach can be made on these, and you will get a very definite amount of good resulting from the surgical approach. If you see no improvement on blocking the bursa, I like at the same time to infiltrate the joint. Many of these horses that have hock trouble will not show any improvement with blocking the bursa, but after you block the joint you definitely get a horse going nearly or almost completely sound. X-raying the hock to diagnose this condition is sometimes rather confusing. I know people will argue, and I know in my own mind that I am not, in many cases, sure whether I have joint lesions or not, from an x-ray. However, when they become pronounced enough so that you have erosion and calcification in the joint, there can be no question about it.

Oftentimes, when this has gone that far, the prognosis on the individual is poor. In most horses that have hock trouble, there is always a secondary soreness in the muscles of the tro-

chanteric bursa, and myositis in the middle gluteus, running from the bursa up towards the loin.

With stifle lameness, the animals travel flat and on the outside of the foot. They will step short and rotate the leg outward. They tend to carry the leg without bending the stifle joint but do not jab the toe as in hock trouble.

However, to me, a horse with stifle trouble has a particular hobbling gait. This is hard for me to explain, but I think they have a hopping sensation, rather than a jabbing of the toe, when they go. It is due, in my estimation, to lack of flexion and underextension of the joint. In other words, that joint does not work freely. There is soreness there. They try to get along with it, going as stiff-gaited as they can. They get a definite type of hopping sensation.

In stifle lameness, the soreness or the myositis you see is higher in the back than it is with hock soreness. It will be higher in the loin area. I think the symptoms run quite characteristic; with hock soreness, you get it in the trochanteric bursa and middle gluteal, while in stifle soreness you get it more in the loin area.

A lot of horses that we see with stifle trouble are young horses, two- and three-year-olds. One of our common causes of bringing on this type of lameness is bringing them on too quickly. They overdo with colts. Whether it is a fatigue factor, or whether they aren't quite properly developed and finally the muscles become tired and fatigued, and they do not handle the joint properly.

A stifle-sore horse, say a colt, is being trained in the South. You put him on a truck and ship him to one of the tracks in New York State. Usually he will come off the truck fairly good. You walk him the next day, and he will walk fairly sound. You start jogging him and he is lamer than a dog. I know other practitioners to whom I have talked have expressed the same opinion, that after moving or trucking, a stifle-sore horse, especially, will show increased soreness.

Primary hip lameness does occur but is usually secondary to stifle, hock and ankle. Primary hip lameness is related to severe injuries or cramping of the muscles associated with tying up.

Another thing that oftentimes causes hip and loin soreness is kidney and bladder disorders. This is something I will not discuss to any extent because Dr. O'Connor is going to give us some ideas on this.

I think there is a proper technic in examining the trochanteric area for soreness. First of all, you want to know your horse to some extent. When you do examine him, you want to be close to him, behind. Start with both hands on the tuber coxa on each side, and put a medium amount of pressure on the tuber coxae to see how your horse is going to react, whether he is a flighty

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horse or whether he is one that is just going to be docile about it, and let you really examine him.

The next thing is leave one hand on the tuber coxa, with the other hand go down to the bursa; put constant pressure from one side to the other and see whether you get any reflex in bearing away from that part. Reverse the side, go to the left tuber coxa and the right bursa. After you have done this, if you see the horse fades one side or the other, put constant pressure on the bursa and see if you get the same type of fading away with it. If you take a little time in this particular examination, oftentimes you can pick out a horse sore in the bursa and help yourself a lot in treating this individual.

As far as treatment in the hock joint, a proper diagnosis is a must. With a surface lesion involving the cunean bursa resection of the cunean tendon is the best. For relief therapy to finish a season injecting the bursa with a combination of Xylcaine, steroid and Sarapin works well. In handling a joint lesion the systemic use of steroids are well as the local injection of the joint tends to remove the pain so the animal will flex the joint normally. The hip in the area of the trochanteric bursa must be treated also as there is always secondary soreness in that area. Local infiltrations of the same material used in the cunean bursa does a good job. After repeated injections in the hip use of an internal blister (Lugol's Iodine or McKay's formula) often will give better results.

In treating the stifle, we have to deal with two things. One is joint soreness involving the lateral collateral ligament of the joint. This is associated with fibular defects and a displacement of the proximal end of the fibula. The use of a sclerosing between the fibula and tibia will tie the fibula down so the lateral ligament of the joint has a stable attachment and will not stretch. Also local injections of an irritating agent over the ligament will tend to strengthen and tighten the ligament. The other condition in the stifle is the upward luxation of the patella which comes from an injury, poor conformation or improper conditioning of the younger horses. In the last case the colts are under-developed in the extensor muscles of the stifles and they have weak sloppy stifles. In treating this last group, use of irritants to shorten the patellar ligaments and hold the patella down where it belongs is necessary. Also plenty of slow work miles is a must to help these colts develop properly. Often times as these horses grow older they tend to out grow this situation. Injection of irritants over the patellar ligaments and in the muscles above the stifles will often show marked improvement in a short period of time. Injections should be repeated at two weeks intervals until the desired results are gotten. It is very important after horses are injected with Lugols or McKay's solution that they be exercised well. If they are let to stand around they will stiffen and become

very sore. Local injections of more than 2 cc. of material in one spot is not good. Multiple injections of small doses are the best. These injections are placed deep in the muscle with an one and one-half inch needle. In some cases a combination of Sarapin Xylcaine and steroid are used for temporary relief in removing soreness in the muscles above the stifle and over the patellar ligaments. In horses having stifle trouble over a long period where the patella is riding too high, cutting the medial patellar ligament at the distal end as close to the tibia as possible will give dramatic results.

DR. JAMES T. O'CONNOR: Mr. Chairman and Gentlemen: When Dr. Woodcock asked me to speak to this group, I chose medication of the standardbred, the old versus the new. At that time I had no idea that we would have such a session as we had Monday on the drug problem.

However, I would like to speak of this problem from the standpoint of the harness horse. That is a standardbred panel.

Harness racing has been and is being called America's fastest growing sport. We, I am afraid, must soon replace the word "sport" with the word "business." I myself make this observation, and it is my personal thought.

Twenty-two years ago I began my apprenticeship in what was once a sport and have since then spent many thinking hours on the magnificent standardbred, through its growth in popularity up to the present day.

I witnessed the sport before the day of the saliva and urine testing, and also followed with intense interest the principles of medication of those earlier days, up to and including our present status.

I am sorry to say that our older men in the profession are passing on one by one, as are our older type caretaker, which is a word we never hear today.

If at any time in this paper I seem critical, I hope it will be accepted in the light that I intend, and that is of a constructive nature. If the shoe doesn't fit the foot, then it is up to the individual himself.

We are today medicating horses, many of which have not been properly diagnosed, due to the frame of mind of the trainer and even the owner who bring excessive demands, with the almighty business outlook standing before the welfare of his animal. This high speed of the business steps up our metabolism so that we cannot, often, given the proper time to the individual. Many of us are pressured into medicating horses by these gentlemen whose knowledge of the drug of choice is frightening. Pecuniary reasons augment this trend. It is due to the stepped-up racing conditions of the time.

As practitioners and clinicians we must not overlook the fact that we as horse doctors deal with objective symptoms, and those

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alone. Many conditions that we treat are difficult to name, as such. It would be wishful thinking to have one drug for one or a group of named disorders, on down the line, of conditions encountered.

We certainly must intensify our diagnostic approach in order to honestly medicate these racing animals. We have unknowingly accepted medicaments that have been falling by the wayside daily, only to be replaced by other first cousins from the same parent stock.

In the past ten years, the corticosteroids have been greatly used. Beginning with cortisone and hydrocortisone, as a starter having only a few depletionary problems, such as potassium loss, we have progressed to stronger cousin drugs in orderly fashion, so to speak, so that now we not only have to contend with the sodium and potassium problem, we have to worry about additional losses such as calcium, phosphorus and other elemental necessities so needed.

These problems are important to you as standardbred practitioners, when realizing the exercise warm-ups, and the miles and miles of preparatory work it takes to make a 2-minute horse. You also know that drugs, through lay channels, are being given to race animals with a so-called do-it-yourself-kit attitude that are proving detrimental to the longevity of top performance.

It means this: We must watch the electrolyte balance and watch it carefully. We must watch their hormonal balance and, also, any factor or drug that would upset their autonomic nervous system directly or indirectly, as these three systems govern the performance of their entire body.

The much discussed anti-inflammatory drugs are fine, as are the steroids and vitamins, and have done much, to our way of thinking, in changing the course of disease. However, they must not promiscuously get near that Texas broom of Dan Anderson's, who spoke of sweeping much under the proverbial table, because they will then be adversely spoken of, I am sure.

What has happened to our older larger animal drugs, aconite, nux vomica, gentian, belladonna, lobelia, stramonium, and many others that were used and proven for so many years? Have present day Pharmacologist and modern thinkers removed them from us for what they won't do, and yet failed to tell us what they did and, for some of us, what they are still doing? Is it right to have poly-pharmaceutical formulas from five or six different sources, all claiming resulting cures, yet all radically different in their own proportions? Do supplemental dealers and scientific minds believe they are going to improve or replace nature and its natural feeds with the ever increasing volume of addictives without doing critical research on the race horse?

The answers to these thoughts are obvious. Gentlemen, it boils down to this: They — I am speaking of the people we are

dealing with — must come to us, the veterinarians in the standardbred field, for help. The veterinarian some day will gain his rightful place amongst these businessmen and sportsmen interested in standardbreds. That will depend on us, for we will naturally all have to pull together for success.

I am going to say just a few words on a condition we are now having in the standardbred, that I honestly feel is going to come to the forefront. We have a myositis syndrome, not "tying up" — I am not getting into that field — in which we have a symptom complex which we cannot pinpoint the actual trouble. However, we get this crouching, drawn up approach, where a normal-gaited type animal will start to change strides. These animals usually are immediately taken to the blacksmith shop and re-balanced for one supposed reason or another. In all this upsetting of balance, we seem to get further and further from the point of original trouble.

An affected runner acts as though he is running with a jack rabbit type gait. He does not extend himself forward, up underneath the body with his hind legs. In the last four to five years, along with some thoroughbred practitioners in New England, I have screened a great many urine samples from these animals with this type of symptom.

The laboratory report will come back as to the classification, mainly beta hemolytic Strepococcus, or another laboratory will call it Micrococcus viridans, hemolytic type, or hemolytic viridans type, whatever their classification is. Ninety-nine-and-so per cent of these animals run a very high alkaline urine. The seat of this organism is in the M.M. of the bladder. We think of the ph of the herbivore as being more or less alkaline. However, the race horse does run a little to the acid side.

These organisms in the last five years, according to the sensitivity work done on them, have changed their pattern. First penicillin was the choice; then they went through the whole antibiotic groups through the mycins, and now they are back to penicillin, and they are getting stronger by resistance. Animals, under treatment, are set back in their racing because the antibiotic therapy, usually is given for a week.

The factor I wanted to bring out was this: We have been misled, or we have overlooked the fact that, to acidify the horse, acid sodium phosphate is not the drug of choice. The herbivorous animal excretes most phosphorus from the intestines. This is a very poor acidifying drug for the horse. It would be much better to use an acid type salt as an ammonium chloride potassium nitrate combination.

We have found that by leaving the antibiotics out in selected cases, we can quickly help affected animals by simply changing the urine ph to a range of 5-5.5, with the combined diuretic affect of acid salts, and the change from alkaline to acid urine.

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With a sterile protein type of treatment — I do not believe this is empirical but some people have their doubts about it — we have been able to pick up a slight, natural response or natural protection against this organism in infected animals. In animals last season, we segregated and tried just this one type treatment without an antibiotic. We do believe we are getting results. We are in that frame of mind now that we must change the PH and increase the natural specific resistance to the organism. I believe I have taken enough panel time. Afterwards, if we have to answer questions, I will be glad to, if you have time. [Applause]

PROBLEMS IN A PONY PRACTICE

R. S. Lundvall, D.V.M.

Shetlands do present some rather peculiar problems, and not only the Shetlands themselves, but the owners often present a little more peculiar problem. Some of these animals are owned and kept in towns and cities, or suburbs, and the people involved have not had experience in handling livestock.

At the Shetland Pony Congress a month or so ago it was amazing to find that people could own and try to raise animals with as little knowledge of animal husbandry as they had. The questions asked of the panel were principally those dealing with husbandry problems.

As you all know, the Shetland ponies are increasing in numbers. In the clinics at I.S.U. approximately one-fourth of the cases are Shetland ponies. The problems encountered are relatively similar to those seen in larger breeds, with some exceptions, especially relatively more breeding problems and more fractures. Also, less wire cuts are seen, but more nutritional problems. Nutritional imbalances are probably one of the more serious with which we have to deal. Finally, laminitis is very common, and this could be classed under nutritional problems in most instances.

Certain facets of pony practice are a little bit different. Pregnancy examination, for example, is called for rather routinely by a lot of the owners. If one has a large hand and tries to perform a rectal palpation on a small mare, considerable difficulty may be encountered. But with good lubrication and plenty of time, one can palpate most mares. If one cannot possibly get the hand through the anal sphincter, about the only thing to do is to use the serum or blood test. It is relatively accurate, and attempts to improve the accuracy are being made. It is quite reliable during certain periods of the pregnancy.

The breeding problems, as such, that we see are usually due to ignorance on the part of the owner. Again going back to nutrition, Shetland pony people often believe that Shetland ponies do not need anything but cornstalks and grass. "A quick way to make money is to buy a couple of mares, turn them out to pasture, and breed them and sell the colts for $200 next spring". This does not occur very often.

So what we run into is, first, correcting the nutrition. Then see that the animals get plenty of exercise. Then correcting the stallion management. Again, the ignorance of some owners is surprising. They will turn a stallion out with a mare for a day and decide the animal should have been bred, and that the mare should have a colt next spring. Six months later, some mares are noted to be in heat, and upon examination, all of them in the

band are barren. Breeding problems and management problems go hand in hand, and it is primarily owner education that is necessary.

There are a few things that are done a little bit differently in Shetlands than in larger breeds. There was a nice panel on colics yesterday. Unfortunately, in many cases in the Shetland it is difficult to perform a rectal palpation and determine the exact organ involved and the exact type of colic. I will disagree, in one way, with the percentages given as to cause. We feel that better than 50 per cent of the colics in Shetlands are due to verminous aneurysms. Of the fatal ones that we see in the clinics — we may not see a representative sample — nearly 100 percent are found to be due originally to verminous aneurysms. We find that most of the strangulations, intussusceptions, volvuli we see are primarily due to aneurysms along the middle mesenteric branch. Apparently, often there is enough impediment to the blood supply to cause an irritation of the gut, too much motion results anteriorly, and not enough posteriorly.

One other common cause of colic in colts is impaction. We feed primarily alfalfa hay in our area. When animals are on alfalfa, we do not feel that we have too much trouble. When the colts are turned out on cornstalks or rough grass, trouble occurs in colts just beginning to eat, and up to a year and one-half of age.

Obviously, in the smaller animals one cannot get a hand or arm into the rectum to establish a diagnosis, or an exact diagnosis. We know they have an obstruction of some kind. So, our procedure has been to administer a quart or two of mineral oil, wait about five hours, and, if the mineral oil is not coming through, to perform a laparotomy. Animals presented with a suitable history are operated upon immediately. Three or four methods were described yesterday, and all of them are of value. I personally like to enter the left flank, and have the animal down in lateral recumbency with a heavy dose of tranquilizer and use a local. One may use the midline or paramedian, site, or enter the right flank. Most of the impactions will be found to be masses of ingested coarse hay or grass about tennis ball size, occasionally a little smaller.

We find that our percentage of success rises remarkably with speed in the surgical procedure. If there is an impaction, we never pull, if possible, the affected portion of the gut to the outside. Also, we make it a point not to insert the hand more than once. In other words, one puts the left or right hand into the peritoneal cavity, grasps the obstruction, and breaks it down. We find that the animal usually does not need any antibiotic therapy following surgery, if proper antiseptic procedures are followed.

In an institution with students in attendance, the students are very anxious to palpate the peritoneal cavity and try to

determine the obstruction, if one is present. We find that if we let one or two students put their hands in, no matter how carefully they scrub, we will have trouble with peritonitis. In other words, repeated insertion of the hand introduces a more massive load of air-borne and skin-borne contaminants.

Again, I will agree with the gentlemen yesterday who stated that plunging the hand through the muscles, similar to the procedure used in spraying cattle, is the method of choice. The midline incision is fine on the smaller colts, as one can expose the ventral portion of the intestinal tract. However, with the midline or paramedian site, there is some danger of a hernia developing, though I never noted one.

One other thing seems to increase the survival rate. This is the administration of normal electrolyte solution prior, during, and after the surgery. It is remarkable the effect that electrolytes have on the pulse in these toxic animals. Part of the toxic symptoms are due to dehydration, A part of the beneficial effects of electrolyte solutions may be due to reversing the dehydration. Part is the detoxifying action by the dextrose fraction. We did use blood transfusions for some time. The only difficulty was incompatability, even with the dam as the donor. The last animal I operated upon, I thought needed blood. I bled the dam and gave the colt 150 cc intraveneously; I turned around to say something and the animal was dead. Maybe it would have died anyway. If the blood is compatible, it is fine; if it isn't, you are in trouble.

On the other types of colics we are pretty conservative and usually stick to mineral oil. We feel that oil, to say the least, is innocuous. Unless it gets into the lungs, it does not do any particular harm. Injections and oral cathartics are used occasionally, but preferably we use oil and Epsom salts.

Passing a stomach tube in the Shetland may be a little difficult. One cannot buy a stiff enough tube that is small enough in diameter. We use a one-half inch outside diameter automotive brake hose, and weld a piece of standard size stomach tube to it for an adaptor. A horse catheter may be used, but it is disconcerting to be passing the catheter and all at once see the tip coming out the other nostril. This is a lot less embarrassing than administering a bolus, and then have the animal suddenly show symptoms of choke. That gets to be a little rugged. We just do not use a balling gun or boluses on Shetland ponies. They may have a large mouth and big teeth, but have an awfully small esophagus, and boluses and capsules lodge very easily. A dose syringe is used in some instances to administer small volumes.

Tercapsol is used very frequently in colics, and is a very good product. Again, we shy away from capsules, as we have seen cases in which the top of the gelatin capsule has dissolved first

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after lodging in the esophagus, and the Tercapsol has caused considerable necrosis.

We have a barn man that has been with us for twenty-five years. The first thing he asks when a client brings in a Shetland pony is, "Is it foundered?". His opinion is that ponies either are foundered, have had founder, or are going to be foundered. I am not so sure that he isn't right, especially with the one-pony owner.

As to the treatment of laminitis, if one would ask 100 opinions, one would get 100 answers. If seen in the early stages, cortisone preparations and antihistamines may be of some value. Unfortunately, a certain percentage will turn up their toes rather suddenly with the intraveneous use of antihistamines. Cortisone preparations in milder cases has proven to be of some benefit, but we still prefer soaking the feet in cold water along with emptying the digestive tract and correcting other causes if known.

Chronic laminitis with rotation of the third phalanx and elongated hooves is difficult to deal with, to say the least. We trim the feet to as nearly normal proportions as we can. If the hooves are not too long and grossly deformed, we have resorted to grooving the hoof wall, both longitudinal and circular grooves, plus median neurectomies. On occasion a valuable brood mare has, let's say, suffered a median neurectomy under our hands. Actually, this does have some benefit in certain cases. We think some of these brood mares that do not exercise properly will have breeding problems from this cause. If we can relieve some of the pain in the foot, the animal will exercise and probably won't become quite as fat, and have a much better chance to bear a foal. One thing about grooving the hooves, one can stall the owner off for six months, because it will take that long, and probably a little longer for the grooves in the hooves to grow to the ground. If necessary this can be repeated. Grooving does, in some cases, allow the hooves to expand and assume a more nearly normal shape.

The second most common lameness in the Shetland pony is luxation, or subluxation, of the patella. It is probably a breed characteristic, and I think it is becoming more prevalent because of the conformation the Shetland pony people are striving for — straight-hocked animals with fine bone.

Along with conformation, nutrition does play a part in chronic luxation and acute luxation, or some that might be termed subluxations. Colts show up with this at approximately six months of age. The owner will state that the pony was cast in a stall, couldn't get up, and the leg was stiff. Eventually straightened out, the pony walks with a peculiar gait. If we quiz the owner carefully, we will often find that he has not fed the animals properly. In young colts such as this, a hemoglobin determination will usually indicate the animal is anemic. A radiograph of the stifle will show very poor bone quality, and that

the tibial tuberosity has pulled loose. A certain percentage of these can be helped by stall rest, freeing them of parasites, and improving the nutritional state. We usually add steamed bone meal to the ration. The excess calcium and phosphorus is eliminated without any difficulty. Injections of a counter-irritant around the patellar attachments, or, sometimes, an external blister may be successful.

The older animals do not respond to this type of treatment. Frankly, I have a tendency to be in a hurry and section the medial patellar ligament immediately in most cases. A small skin incision, not over one-half inch, is made; a curved scissors is used to establish a tract under the medial ligament, and the ligament is severed with scissors. A bistoury is seldom used to sever the ligament. All of these are done standing. The weight should be on the leg, to keep the ligament taut so it can be distinguished from the rest of the tissues. Using a scissor, if the animal jumps, the skin will not be cut. One suture is placed through the skin incision. A certain amount of exercise is necessary. The sutures are removed in about five days.

If one should be so unfortunate as to come out with joint fluid, carefully ignore it, and suture the skin. That is, provided the preparations have been aseptic. The joint capsule around the patella does pouch down between the ligaments to some extent. This will vary with individual animals. It is rather uncommon, but does happen on occasion, that one will penetrate the joint capsule. This apparently does no harm, provided infection is not introduced. We allow a certain number of students to perform this procedure, and they will penetrate the joint capsule on occasion. Apparently, it usually results from turning the bistoury or point of the scissors upwards. The bistoury or scissors should be directed parallel to the floor or slightly downward.

For a month or so following sectioning to the ligament, the animals do have a slight "looseness" of the stifle joint. One can see the joint move in and out, but this gradually disappears. Quite a few animals are being shown in both harness pony classes and at halter after having had the medial patellar ligament sectioned.

Parasite control is a big headache in Shetland practice. We have not found any Shetland pony band that is free of internal parasites. Again, this reflects upon the management practices. Some raise Shetlands just like pigs; in other words they are not a band of ponies, they are a herd. The animals are crowded together in small pens, the hay is thrown on the ground, and also the grain ration; the colts run with the mares and are also fed on the ground. If fecals are run on the animals, they are found to be absolutely loaded with parasite eggs. If one happens to die, and it is posted, it will have all of the parasites known to man or beast.

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Unfortunately, some of the owners are not equipped or do not have enough area to scatter out these animals, and it is a constant battle keeping parasites in check. The owners are pretty well confused on parasite control. It takes a considerable amount of explanation to try and get them to understand that worming the animals periodically will usually not solve the problem. They have to be shown the facts of life, in that control goes back to basic animal husbandry and sanitation. If we can get this point across, we feel we have accomplished something.

As to the actual mechanics of treatment, we have on occasion run into trouble with certain vermifuges. I do not know quite how to approach it, but the fact remains that phenothiazine is the agent of choice, as nearly as we can determine, for the large strongyles. Unfortunately, if an animal is anemic and a full therapeautic dose of this product is used, one will further complicate matters because phenothiazine in itself will tend to destroy the red blood cells. Some animals also will show an idiosyncrasy toward phenothiazine. In the abnormal animal, one that is anemic, either is quite serious.

Hemoglobin determinations were made on several animals from a band of ponies that became ill within 48 hours after a full therapeautic dose of phenothiazine had been given. The laboratory reported levels from 3.5 to 5 Gm. hemoglobin per 100 ml whole blood. We attempted to treat these animals with intraveneous administration of whole blood. The results were inconclusive as to the benefits. Talking to a pathologist about this, he said, "I wish you would keep track of the surviving animals, and take a look at them in six months to see what happens to the liver." The broken down blood, in his opinion, would cause liver damage at a later date.

In treating ponies that are not doing well, or, as the owner says, "are not doing right, Doc, I want them wormed," we make it a policy to run a hemoglobin determination before we administer phenothiazine. If we find the level low, we either cut the dose of phenothiazine in half, or use something else. This may not be very efficient as to removing the parasites, but it is not very efficient in producing death of the animals either.

Considerable quantities of the piperazine compounds have been used, and are very efficient in removing roundworms. We do not believe these compounds are very toxic. Unfortunately, none are very effective against the large strongyles. These preparations will eliminate most of the small species of strongyles. Quite often the piperazine preparations are added to phenothiazine both in less than full therapeautic doses. This combination seems to be quite effective and has the advantage of enhancing the anthelminthic toxicity without increasing the toxicity to the animal. Or, one of the piperazine compounds or preparations is administered first, and a week later, the phenothiazine.

A peculiar parasite in Shetlands is found fairly often, and occasionally in other breds. This is S. westeri — a small strongyle. As far as we can determine, it is not affected by any drug not capable of destroying the animal. It is ordinarily not a serious problem, except in certain bands of animals in which the infestation is so heavy that it will produce a diarrhea in colts. We have been using diathiazine for a comparable species in the dog, so, we transposed the results and hoped for the best in Shetlands. About all that happened was that the stalls were stained blue by the feces. Some small decrease in the numbers of parasite ova was noted. Ordinarily, adult animals are not visibly affected by this parasite.

Castrations in Shetlands are no different than in larger breeds, except that cryptorchids are increasing in incidence. Someone had an article in a trade journal a month or so ago stating that Shetland ponies' testicles descend later than in other breeds. Possibly, this is true in certain strains of ponies. Unfortunately, the sire and dam both are usually descended from cyrptorchids — this is the only difference. Some of the better show lines of Shetlands are descended from unilateral cryptorchids, and if you try to talk the owner into not using a unilateral cryptorchid as a stallion, you are probably wasting both parties time.

Unfortunately, owners receive a lot of advice, gratuitous and otherwise, and have come up with the startling fact that unilateral cryptorchids are usually fertile; and more masculine, in some cases, than normal stallions. Then they are determined to use these animals if they are good type individuals, otherwise. The factor is definitely inheritable, and I feel strongly about it. Every time we see a cryptorchid, or a high-flanker, we castrate him if the owner will allow it. If the owner doesn't allow it and wants something done to bring the testicle down into the scrotum — massage, hormone injections, surgery — we politely tell him to peddle his wares someplace else. We are doing the pony breeders a disservice and we are cutting our own throats if we let these people use cryptorchids for breeding stock.

Strangles is very prevalent in Shetland ponies. Some mention has previously been made of abscesses. The last few years we have stopped treating strangles medicinally. Experience has shown that the use of antibiotics will definitely keep the abscess from pointing, and it will recede a little. However, as soon as the antibiotics are stopped, in a large percentage of cases the abscesses will recur. We thought we had the answer when penicillin first was available. The Streptococcus involved was apparently very susceptible to the drug. We kept the animals in the clinics, injected them daily with penicillin for six, eight, or ten days. Things went along fine. We sent them home cured, and two weeks later the animals would be back with the same complaint.

Unless the animal develops pneumonia, or threatens to do

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so, we now recommend hot-packing, incising the abscess, and letting nature take its course. If pneumonia does show up, it is often incurable because of multiple lung abscesses. Oxygen therapy may be of value, and is a little easier to administer than in the larger breeds.

Fungus infections seem to be increasing in incidence. A mycologist who is interested in fungus problems in horses has run a survey of a large number of animals with skin conditions. It is surprising the number of fungi that are isolated. Most respond to sanitation plus specific treatment of the individual animal, or individual lesion.

Thank you, gentlemen.

CHAIRMAN WOODCOCK: Thank you very much, Dr. Lundvall.

Are there questions?

DR. O. R. ADAMS [Fort Collins, Colo.]: I think this is the group you ought to start calling patellar subluxation, fixation of the patella. A lot of literature refers to lateral luxation, which is an entirely different condition, very rare, and the animals cannot bear any weight. Do you agree with that?

DR. LUNDVALL: Absolutely. The only reason I used "luxation" is because it is a common term. It is upward fixation, you are correct. This same article I referred to, not too long ago, had something about these patellar fixations, in that they were incurable. I will disagree violently with that. They are incurable if you let them go, because I think, as Dr. Adams is trying to bring out, there is a flattening of the trochlea, as it goes on.

DR. ADAMS: In my opinion, there is only one cure. I have been arguing about it a lot. You should quit extending the leg.

DR. LUNDVALL: Do you sever the medial patellar ligament?

DR. ADAMS: Always.

DR. LUNDVALL: We find the pull is enough in colts on poor rations, that you can actually move the tibial tuberosity with your finger; in other words, the attachments. You can get your finger under it and move it, the bone is that poor quality in some cases.

We usually feed bone meal. They need it. It does do some good. Another thing I will mention: Usually I find on these poorer grade colts it is a rather futile proposition to clip or sever one medial ligament. I usually do it bilateral because it commonly—let's not say all of the time — but at least 50 per cent of the animals have poor conformation. I sever one medial patellar ligament. Three weeks later the animal is back, and I have to sever the other one.

THE DIAGNOSIS AND SIGNIFICANCE OF CARDIAC ARRHYTHMIAS

D. K. Detweiler, D.V.M.

Thank you very much. Ladies and Gentlemen: After I was asked to talk on cardiac arrhythmias in the horse, I became curious about when veterinarians first were aware that horses had cardiac arrhythmias. I am not sure I have gotten back to the first report of this.

The first one I could find was just about 100 years ago, in 1858, in one of the earliest textbooks written in veterinary medicine. It was written by Falke, the city veterinarian of Jena, in Europe, who was apparently at the end of his professional life, from the introduction of his book. He tabulated his many experiences as city veterinarian according to body systems and diseases which he found.

Under his discussion of heart disease, he listed a number of heart diseases in horses, mostly based on postmortem evidence, and mentioned that in these horses which had heart disease, such things as myocardial degeneration, myocarditis, rupture of the heart and so on, he had noted, in many instances, cardiac irregularities during his clinical or premortem examination.

Thirty years later I think the first actual case report, in a horse, of cardiac arrhythmia *per se* was published by another European veterinarian, Vogel. The original report is not available to me. I have read it in abstract. This apparently was an instance of a horse probably with myocarditis which had developed, initially, in complete heart block, that went on to complete heart block, and the animal died.

However, the first significant publication on cardiac arrhythmias in horses was much later than that, in 1924, when Johann Norr was at the Veterinary School in Giessen. He was actually, I would say, the father of veterinary electrocardiography. In 1913 he obtained one of the first string galvanometers and began to study horses. Nine years later he reported on 100 cases of cardiac arrhythmias in the horse.

Since that time there has not been much of significance actually published on cardiac arrhythmias in the horse, until a few years ago when Brooijmans in Utrecht published a monograph on clinical electrocardiography in horses and cattle. This, I think, brings us up to date with respect to the history.

I will say that I fear that we do not know much more about the significance of cardiac arrhythmias in the horse in almost 1960 than Johann Norr knew back in 1924. There have been very few significant studies done since his.

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Before discussing the arrhythmias themselves, I would like to talk for a moment about diagnostic methods. The methods for examining the cardiac rhythm are apparent to all: Palpation of the arterial pulse; observation or palpation of the jugular pulse; auscultation of the heart; palpation of the precordial region; electrocardiography. These are the methods we can use in evaluating the pulse rhythm.

In making an examination of the pulse, I think it is important to monitor the pulse rhythm for a given period of time.

The next decision to make is how long must we do this? Certain arrhythmias only occur occasionally. Some of them only occur very occasionally. Actually, I think it would be well to monitor 1,000 heart beats but this is not practical; it would take too long.

Our procedure is to attempt to monitor, as continuously as possible, the pulse rhythm or the heart rhythm for a period of between five and ten minutes, hoping that during that time, if there is an arrhythmia present, we will detect it, feeling that any arrhythmia that occurs less frequently than that is probably not too significant.

One could listen to the heart for ten minutes. This is often inconvenient; the animal might get restless, and so on. We try to accomplish this by the following method: As soon as the horse is approached, if he is fairly quiet, not tossing his head, we begin to take the arterial pulse at the maxillary artery, and take this for about two minutes.

During this time you can determine whether or not it is easy to see the jugular pulse at the thoracic inlet. If this is easily observable in the animal, then for the next half minute or so, just observe that. The next thing we usually do is begin auscultation. If the animal has been restive to begin with, since it is easier to hear the heart than palpate the pulse when the animal is moving around, tossing its head, and so on, we will start out with auscultation.

Then we auscult the three important areas on the left side; at the apex, the fourth intercostal space and the third under the foreleg; pass around to the right and auscult under the foreleg, listening for murmurs and taking about three minutes to monitor the rhythm to determine whether or not any arrhythmias are occurring.

Finally, if we have started out with arterial palpation, gone back and listened to the heart, then we finished off with several minutes of just observing. If the horse is now quiet and we can observe the jugular pulse, we observe that for some minutes. If in the history of the animal there has been an arrhythmia and nothing has been detected during the ten-minute period of observation, another way of getting at this is to tie the horse in a position, so you can observe the jugular pulse. It is not clearly

visible, as you know, in all animals. Part of this depends on the position in which they carry their head. We get out of the stall and place the animal where it cannot see us and be disturbed by our presence, and we watch the jugular pulse to see if any arrhythmias occur.

Another useful device is to observe the pulse as it slows down from either exercise or excitement, because certain types of arrhythmias, for example, ventricular extrasystoles, may only show up at this time. A good way to do is to have the handler shake up the animal so that it gets excited. We like to get the heart rate above 100 for this purpose, quiet him down quickly, and auscult as the heart slows down; observe how long it takes to slow down and if any arrhythmias develop before the resting rate is attained. If arrhythmias occur during this period, we generally think this is evidence of heart disease.

Finally, we use the so-called exercise test in which the horses are exercised within the limits of their tolerance, and the cardiac rhythm monitored with the electrocardiogram continuously for about eight minutes.

Finally, stay with the animal for from fifteen to thirty minutes to see if any arrhythmias develop even some time after exercise. If this much care is taken, it is surprising the number of horses that have arrhythmias.

The next matter concerns the significance of these cardiac irregularities. As I said, this is still very controversial, and I think in recent years we have not learned much.

Before getting into the specific arrhythmias themselves, I would like to go over some of the basic physiology of the pacemaker and the rhythm of the heart.

There are a couple of very brief textbooks that can be read for this purpose, that I thought I might mention. Actually, they are both so short that you can read them in one evening, and they contain all the fundamentals one needs to know. The one book is called *Electrocardiology* by Schamroth. It is a very basic book of about 120 pages, mostly diagrams, and it really simplifies basic electrocardiography.

Another book which talks about hemodynamics in an applied way, that I find very useful, is by Wilde. This book is called *The Fundamentals of Cardiology*, published by C. C. Thomas. Another very brief book, mostly diagrammatic, that one can cover in a couple of evenings and contains the basic information you need.

I want to emphasize that I do not think there is any specific cardiology of the dog or of the horse or any other animal. These basic principles, in general, with slight modifications, apply to all species, and this is the place to start.

[Slide] In discussing the arrhythmias, I would like to review the rhythm regulation in the heart. Here is the pacemaker, the sinoatrial node. In the horse the sinoatrial node, or SA node,

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is much larger than depicted here. We think it actually extends half-way down the atrium, between the two venae cava. It is a large, horse-shaped structure. This needs verification. It seems to be quite a large structure in the horse.

This is the atrioventricular (AV) node, the beginning of the conduction system, carrying the impulse into the ventricle.

During the beat of the heart, the pacemaker initiates the heart beat. It spreads it in a wave-like fashion over the two atria, and encounters the AV node.

Here it is held up for a brief time. During this period the P wave is inscribed. Then there is a period of delay in which this impulse does not pass in the ventricles. This is during the straight segment of the so-called P-R interval. Then the impulse passes very rapidly into the heart. This is the electrically silent period. We have no evidence, from the electrocardiogram, of this movement of the impulse through the specialized conduction tissue. Then it leaves, rather simultaneously, to activate the ventricular mass. Since that activation is not entirely synchronous, we get a complex series of waves, the QRS complex.

Then all during systole, the tracing returns to the isopotential line. The repolarization is also not synchronous so we get another wave, the T wave. That is the end of contraction.

[Slide] This shows a schematic diagram of the conduction system in a horse's heart. The conduction system is very extensive, more extensive in the equine heart than in the dog or the human or monkey, et cetera.

The SA node, as I said, we think now is quite a large structure, much larger than depicted here. Here is the AV node. You can see the branches of the conduction system going out through the ventricular muscle mass.

[Slide] This shows the relationship between the activities of the atria and the ventricles and the electrocardiogram and the pulse wave. This is from a human, but it applies, of course, to the horse as well.

The open square represents the SA node or the pacemaker. The small, black rectangle represents contraction of the atria; the large one, contraction of the ventricles.

In the normal sequence of events, the pacemaker fires off. This is electrically silent. We have no signal that tells us this. The spread of excitation passes over the atria, and we see the inscription of the P wave, as depicted here.

Then there is a short period of delay, as you can see, between these two events, contraction of the atria and ventricles. Then QRS is inscribed, and at that time ventricular contraction begins.

Just look at this diagram a moment because I am going to use it later to describe a certain type of arrhythmia. The upper black rectangle is a trial contraction; the lower is ventricular contraction. The slant of this line, connecting the two, indicates the

duration of time between contraction of the atria and contraction of the ventricles.

[Slide] Just before I come to that, I want to talk about one type of arrhythmia, so-called sinus arrhythmia. I think all of you who have had experience with dogs recognize that they have a very pronounced respiratory sinus arrhythmia. Their heart rate increases abruptly with inspiration and decreases with expiration. If they are at rest, this is very pronounced.

We do not see respiratory arrhythmia in the horse, but they do have a sinus arrhythmia that can be easily detected in the electrocardiogram but is not sufficiently pronounced that you usually notice it through auscultation when they are at rest.

This pacemaker which was depicted in the last slide is not entirely regular, and varies in its activity for reasons I do not entirely understand.

There is another abnormality involving the atria which is called wandering pacemaker. I just mentioned that the pacemaker, the SA node, in the horse we have reason to believe from old work is much more extensive, much larger than in certain other animals.

What we see, as you can if you look at these records here, is that the shape of the P wave will vary in the lectrocardiogram from time to time. For example, this was taken with the lead down in the esophagus. The P wave is inverted. The shape of the P wave in that complex is entirely different from the shape of the P wave in this complex.

Here is another example. Here we have a notched P wave, and the next P wave to it is just a single upright wave. Brooijmans, who has written the most extensive discussion of arrhythmias in the horse, considers this evidence of disease. I do not know. Out of 47 carefully selected horses—that is carefully selected for normalcy of their cardiovascular system—we found 16 that at some time or another showed this abnormality. While it could be associated with disease, I do not believe it is a reliable sign of disease.

The next arrhythmia involving the atria is sinoatrial block. In this arrhythmia, the pacemaker just fails to fire off once in a while, and beats are skipped. Here you see an example of that. Here is a heart beat. Here is the heart sound. Here is an interval of time just double the dominant rate which you see here, during which there is no beat. The atria do not contract; that is, the pacemaker simply failed; neither do the ventricles. This is an uncommon type of skipped beat. We think that it does not necessarily have any clinical significance.

[Slide] The next slide shows another instance of this. Here you see the series of beats going along regularly. The horse misses a beat; neither the atria nor the ventricles contract.

[Slide] The next type of arrhythmia which causes skipped beats is atrioventricular block. This is the type of arrhythmia in which the atria contract as a result of pacemaker activity, and

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the impulse fails to be conducted to the ventricles, with its normal speed. That is, it does not get there as quickly as usual, or it does not get there at all sometimes. There are different types.

Here, then, is depicted a normal situation in which the atria and the ventricles contract, following one another by an interval represented by this slanting line.

In first degree atrioventricular block, the only thing that happens is that the interval between the atrial and the ventricular contraction is somewhat prolonged. You can see this by the change in the slope of this line. In second degree, or partial heart block, usually what we see in the horse is a prolongation, with a change in the slope of this line of the interval between atrial ventricular contraction. Then a beat will be skipped. That is, the conduction does not take place at all, and the ventricles fail to contract. After that, the PR interval, or interval between atrial and ventricular contraction, will become shorter.

Here you see another type in which the PR interval remains constant; that is, the interval between atrial and ventricular contraction remains constant. Despite that, beats are skipped occasionally. This is a very rare type. This occurs in man, sometimes.

Finally, we have the type of atrioventricular block in which the atria are going along at their rhythm but none of the impulses originating in the pacemaker reach the ventricles. The ventricles set up their own pacemaker and beat at their own rhythm.

[Slide] The next slide will show you some examples of this. Here is a record taken before and after exercise in a horse. Here is the PR interval. This indicates the interval of time between atrial contraction and ventricular contraction. Notice its length. With acceleration, when the vagal tone decreases with exercise, this interval should shorten. Look what happened to this horse. Here we have a faster rate, and the PR interval has lengthened. This ordinarily does not happen with exercise. The PR interval should shorten. This abornal prolongation indicates there is some abnormality, perhaps a reflex thing, involving the vagus nerve, or disease of the conduction system itself.

This would then be called incomplete heart block. It is an electrocardiographic diagnosis.

[Slide] This is the kind of thing you hear clinically, in which the horse is just dropping beats: 1, 2, 3, and then he misses a beat; 1, 2, 3, he misses a beat; 1, 2, 3, 4, 5, 6, 7, then he misses a beat. This shows a number of phenomena which are interesting.

We have always thought, or it has been known in the dog, in the cow, and in humans, after the misses beat, the interval between atrial contraction and ventricular contraction is shorter than it was before. The reason for this, as I am going to show in a moment, is when the heart misses a beat, the blood pressure falls, this changes the vagal tone, and a decrease in vagal tone

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results in shortening of the delay between atrial anl ventricular contraction.

If you examine this record, however, you will see here is the PR interval. You can see the length of that. Here is a missed beat. This Pr interval should be shorter; it is not; it is actually longer than the pre-block beat. This is something different in the horse. We have never seen this in any other animal. There are a number of complicated ways one might explain it.

[Slide] Here is an interesting thing that will happen. It will happen in any heart, occasionally in horses. This horse, as you see here, was missing two heart beats. Then notice this bizarre complex here. Here is a ventricular extrasystole.

What happens in any heart, if the impulse from the pacemaker does not reach the ventricle for a given period of time, an extopic pacemaker will be set up in the ventricle, and it will contract on its own. That is what has happened in this complex here.

When the delay in conduction is long enough and the ventricle itself contracts, I think that this is abnormal, at least statistically abnormal. I would be suspicious of the presence of heart disease.

[Slide] Here is a record from a horse with atrioventricular block, in which we ran simultaneous records of blood pressure. This is the control record, before the block developed. You will see there was a blood pressure here of between about 90 (diastolic) and, perhaps, 140 (sylstolic) mm. of mercury. This is the pulse wave; this is the ECG. We ran the recording paper at two speeds. This is the same record, except five times the speed of this one.

Here is where the incomplete heart block, with dropped beats, began. You can see the beats going along here, 1, 2, 3, 4, then misses a beat. Notice what happens to the blood pressure. Here is the missed beat. The systolic pressure here falls down to around 100, or below 100. Then with each heart beat in the series, the systolic pressure, you see, goes right up until it reaches, perhaps, 130. Then, when the beat is skipped, it falls down again, so it is falling down about 30 millimeters of mercury. We have recorded some of these in which the pressure between beats falls 50 mm. of mercury. That is quite a fall in pressure.

It is no wonder, then, that we notice these rather significant changes in the post-block beat of a reflex nature because whenever the blood pressure falls, then the autonomic tone changes; the parasympathetic tone diminishes, and the sympathetic tone increases. This causes rather profound electrocardiographic changes which are perfectly physiological and are not abnormal, do not indicate any heart disease.

[Slide] Some years ago European workers averred that the missing of heart beats in horses was a respiratory-linked phenomenon, just as the respiratory sinus arrhythmia in the dog was associated with respiration; in other words, that horses missed

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heart beats when their vagal tone was high, and that their vagal tone was high at a fixed time in the respiratory cycle.

We have taken a number of records, simultaneous pneumograms and electrocardiograms records, on horses, recording respirations and recording missed beats. Here is one of them. It will be clear to you, I think, if you look at this, that this relationship does not hold. The missed beats are not related to any part of the respiratory cycle, as far as we can see. Here are the missed beats in the electrocardiographic records. This is the simultaneous pneumogram, or respiratory record. We have drawn a line opposite the P wave of the missed beat in each case. If you look at this, the inspiration is down and expiration is up. Here is a missed beat in the middle of inspiration, middle of inspiration, end of inspiration, beginning of expiration, middle of expiration, peak of expration, beginning of expiration, and so on. I think on this record that they are just equally distributed between inspiration and expiration.

[Slide] The last type of block that I have been talking about is incomplete atrioventricular block with dropped beats, a common thing you see clinically.

Brooijmans, the man who has written the treatise on electrocardiography in horses, says it is a sign of disease. There are many horses, as you know, that are in very good condition clinically, still racing despite having missed beats at rest, and they do not show signs of disease. I believe it is a physiological phenomenon in the horse.

If complete heart block occurs, the ventricular complex is very slow, because these are caused by a pacemaker set up in the ventricles and the pacemaker in the ventricles is always slow. It will go down even as low as 10 or 15 in the horse. Here are the P waves occurring at a rather rapid rate, but none of the impulses from the atria are conducted. This always indicates very serious heart difficulty. It is rare. Most of the animals that have it experience repeated so-called Stock-Adams seizures.

Of all the types of blocks I have discussed, this is the one we can certainly say means heart disease. We cannot say, I do not think, that the other types necessarily indicate heart disease in the horse.

[Slide] Before I take up this slide I would like to talk about atrial fibrillation. I talked about this two years ago here, and about some of the ideas we were developing then regarding atrial fibrillation.

In this arrhythmia, the atrial fibers contract asynchronously, instead of as one. Atria fail to contract. Clinically, one can diagnose atrial fibrillation by the presence of a completely chaotic pulse, and tachycardia usually, although this is not entirely reliable. We have seen some cases with actual bradycarlia. One never hears a fourth heart sound because the atria are not contracting.

If the heart rate is fast, up around 100 or 120, and irregular, there will be a pulse deficit. By that I mean if you have someone count the heart beats by auscultation, and someone take the arterial pulse simultaneously, by palpation, the heart rate will exceed the pulse rate. That is because some of the heart beats occur before the heart is filled with blood, so arterial pulse does not result. The term employed for this disparity between heart rate and pulse rate is pulse deficit. The heart sounds will vary in intensity. If there are murmurs present, they will vary, and the pulse will vary also in intensity.

If this constellation of signs is found, you can make this diagnosis clinically. Atrial fibrillation is a very serious sign of disease. Some years ago physicians became aware that individuals had bouts of it occasionally, in the absence of heart disease. In the dog, certainly in our experience, it always means organic heart disease of very severe type.

However, one observation that was in the experimental literature that interested us was this, that, in the fibrillating heart muscle, if you reduce a mass of muscle to some minimal size, it will stop fibrillating, and you cannot induce fibrillation. In other words, there is a relationship between muscle mass and this arrhythmia, experimentally.

Secondly, we were impressed with the fact that in these large hearts one could, with quinidine, always abolish the arrhythmia, whereas in the dog with a small heart we can practically never abolish it. In humans it is about 50-50. A good many times it can be abolished, and sometimes it can't.

About a year ago we tested this hypothesis by including fibrillation in the beef heart, in intact cows, in calves, in dogs, in sheep, and goats. We found that we could induce atrial fibrillation in the cow which would last for a period of several hours up to several weeks. In all of the small hearts it would never last more than a few minutes at the most, and usually we could not induce the arrhythmia at all by this method.

So, on the basis of this type of reasoning, we felt, perhaps in the case of horses, atrial fibrillation is not a sign of as serious underlying heart disease as is true in other species with smaller hearts such as dog or man.

Accordingly, we have treated some nine now, I think, with quinidine, and converted them from fibrillation to normal rhythm. Unfortunately, I do not have at the moment the reports on all of these. Dr. Churchill, for example, told me last night one we converted two years ago won five races this year. I think, actually, this is the only practical thing that we have learned so far in studying heart disease in horses; that is, at least some of them can have this arrhythmia, that we had always thought was a very serious one and meant serious organic heart disease, and it does not necessarily mean that the heart is too badly diseased

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to permit strenuous exercise as in racing. I hasten to add that this is experimental. I would like to see 50 cases converted and followed before we would draw sound conclusions. We intend to keep up with this. I know Dr. Reed has treated some cases. He probably has some experiences he can add to this. In our cases, all of the ones we have followed, several are running and some are winning races.

Here is an example of atrial fibrillation. There are no P waves. The rhythm is irregular. I wanted to show you something that I think is of interest. Here is the first heart sound in this horse, and here is the second. There is no evidence of a murmur here. We could not hear a murmur in the animal. The heart rate was rapid.

[Slide] This shows the animal after conversion. Here is a rather bizarre bifid P wave, in the normal position at least, the QRS and T complex. Notice how the sounds have changed. Here is the first heart sound, and a murmur running right through systole. We thought this horse had mitral insufficiency. Murmurs are much harder to hear in the fibrillating heart than when it is converted to a normal rhythm.

[Slide] Here is another, not arrhythmia; it is a rate change, sinus tachycardia. The rate is very fast, up around 120 or more. This was in a horse that lacked stamina. I have no other report on it. I do not think it necessarily means heart disease. I just show it to show an example of a very rapid rhythm in a resting horse.

[Slide] During conversion of horses from atrial fibrillation to normal rhythm, you see certain bizarre arrhythmias that we have never seen clinically. Here is one. You see these large, repeated waves. Actually, these are very rapid P waves. They are complex because of the nature of the lead.

Here are the ventricular complexes. This we have called atrial tachycardia, extremely fast atrial beat, with a good many of the impulses not reaching the ventricles. I think this occurs as horses are converted from atrial fibrillation to a normal rhythm. We have never seen this spontaneously. It could occur, of course.

[Slide] Here are ventricular extrasystoles. You see them here, these bizarre beats occurring early, usually followed by a pause. In this case, the atria contracted after the beat, and the ventricles were undoubtedly refractory because this beat has occurred so recently in the ventricles. I do not know what significance to attach to an occasional ventricular extrasystole. Clinically, you can diagnose them through auscultation, if you listen to the heart, and suddenly you hear an early beat, unaccompanied by a second sound. The reason for that is the heart contracts before it is filled with blood, and no second sound can occur. This occurs under a variety of clinical conditions, gastrointestinal dis-

turbances, respiratory infections. I think, for the most part, they are not very serious, if only occasional.

[Slide] However, when you see this picture, the situation is different. An extrasystole occurs after each normal beat. You notice the interval between the normal beat and the extrasystole is constant in each case. If extrasystoles occur every other heart beat or, at least, very frequently, I think it usually means heart disease.

This horse had an acute myocarditis accompanying pneumonia.

[Slide] Here is the normal beat. Here is the first heart sound, QRS; second heart sound, following the T wave. Here is the extra beat, and here is a single sound. That can be confusing. I missed this one on auscultation. The reason was, as you know, the horse very often has a third heart sound. When I listened to this heart, that is what I heard. The only thing that aroused my curiosity was this was a much louder sound. That is the reason was ran the ECG. This rate was slow. Actually the dominant rate was rather slow. This was a serious case of heart disease. On the basis of auscultation, as long as he was having his extrasystole occurring immediately after the normal beat, in that sequence, and he did this every time, I could not tell, by means of auscultation that this was not just a normal heart. The rate as determined through auscultation was normal, even though the true rate was double normal, because I could not identify the extrasystole.

A few minutes later this same animal changed a little bit, so there were two normal beats, and then the extrasystole. It then immediately became apparent that we had tachycardia and that something funny was going on.

You notice he did have a normal third heart sound. He had a first heart sound, second heart sound, normal physiological third heart sound.

In the next beat you see his first sound and second sound. Before the third heart sound can occur, he had this loud single sound, due to the extrasystole. The timing of these events is identical. The distances between sound 2 and sound 3 and sound 2 and the extrasystolic sound were almost the same. This shows, I think, the value of doing routine ECG's.

[Slide] Here is a serious arrhythmia, paroxysmal ventricular tachycardia. In this arrhythmia, this is actually a series of extrasystoles. In other words, the ectopic pacemaker has taken over the rhythm of the heart, becoming so rapid that it dominates the atrial pacemaker. Whenever this occurs, I am sure that we are dealing with serious disease.

Now just to summarize briefly. Of the arrhythmias I have talked about, which are the common arrhythmias occurring in the horse, I think the following arrhythmias are usually benign: Sinus arrhythmia which is present in most horses; a wandering pace-

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maker, that is variation in the shape of the P waves; sinoatrial block, or SA block; incomplete AV block with dropped beats—this is the usual type of skipping of the heart beat which we hear; and occasional ventricular extrasystoles.

These five types of arrhythmias we have seen often enough, in the absence of good evidence of heart disease. I do not think we can base a diagnosis of heart disease on them.

On the other hand, I think the following list of arrhythmias usually indicate heart disease. First, atrial fibrillation. I mentioned that we feel that the atrial fibrillation does not mean a serious underlying heart disease, as it does in man and the dog. Nonetheless, I think it means that heart disease is present.

Secondly, certain types of ventricular extrasystoles. The last one I showed you in which there were a couple of ventricular extrasystoles after every beat, part of the time. With frequent extrasystoles, we usually are very suspicious that myocarditis is present. If they are of multifocal origin; you can tell if they are originating from more than one focus, electrocardiographically; if they are, we think this means heart disease. If they are bidirectional—by that I mean the direction of the QRS complexes in the ECG is reversing every extrasystole—we consider this a very dangerous sign.

Third, paroxysmal ventricular tachycardia, the last arrhythmia, and, finally, complete heart block.

Thank you.

CHAIRMAN WOODCOCK: Thank you, Dr. Detweiler.

Are there any questions from the floor?

DR. D. D. DELHANTY [Ithaca, N. Y.]: Occasionally you run across an animal with a history that may go on over a period of several years. Your block or ECG's will reveal quite consistently a wandering pacemaker, a lengthening of the P-R, and these animals progress over a period of time. Under your classification these are benign lesions.

DR. DETWEILER: I am glad you brought that up. All of these phenomena, wandering pacemaker, incomplete heart block, with or without dropped beats, are, in my judgment, the results of elevated vagal tone. There are many things that will do this. Lesions in the heart will do it. So that I think, when you get this picture, if you are taking a series of ECG's in a horse, and you have this sort of picture where it has been present a long time, the severity of it is increasing in the same animals, and so on, this probably is correlated with heart disease.

However, the point I want to make about these arrhythmias is that they, in themselves, I do not think, are reliable signs. I know we find these things more commonly in the aging horse, but they can be caused by simple elevated vagal tone. There are many things which may elevate vagal tone. Lung lesions can do it, certain gastrointestinal disturbances, etc. I do not believe they are

reliable signs of heart disease, and we should base the diagnosis of heart disease on other signs.

Moreover, in our experience, where we tried to carefully select horses for normal cardiovascular systems, we found these changes. I do think the prevalence increases with age, and we might look upon them as something associated with the aging process. In this sense, if aging is abnormal, they are abnormal. I would admit they will be more frequent in diseased hearts. I still cannot consider them reliable signs. This, as you see, is a controversial point. Others, such as Brooijmans do not agree with my point of view.

If you base the diagnosis heart disease on heart block and that sort of thing, there are a great many horses that are very healthy, as far as I am concerned; they are winning races and living a long period of time, and they just show no other evidence of heart disease.

DR. A. R. SKEWES [Union Grove, Wisc.]: Are there any types of anemia that will confuse, when you are listening to a heart, that will make it sound as though it is a diseased heart, and if the anemia is corrected this will then change the type of heart beat that you have?

DR. DETWEILER: Yes. I think any severe anemia, especially in the horse — certainly, this is also true in other animals — will produce systolic murmurs. We think this is due to increase in turbulence. When the anemia is corrected, these murmurs will disappear.

I might say this, in my judgment, in the horse, one cannot base a diagnosis of heart disease on murmurs. Horses have murmurs so frequently, that disappear, and are not related to any visible lesion in the heart, when necropsied. We have abandoned this as a reliable sign of heart disease. If the murmur is intense enough to cause a thrill, I think it is reliable. If the only thing we hear is a murmur in the heart, there is no arrhythmia, no conduction difficulty after the exercise test, if the heart is not enlarged by percussion, we do not call it heart disease. Too many times I have heard a murmur in the evening and have gone back in the morning, and the murmur which was present the day before has disappeared. Murmurs in the horse are very confusing. We certainly need to work on this.

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THE "TYING UP" SYNDROME

A PANEL DISCUSSION

Moderator: B. F. Brennan, V.M.D.

Panel: Robert R. Marshak, D.V.M.

G. H. Keown, D.V.M.

O. R. Adams, D.V.M.

R. L. Lundvall, D.V.M.

E. A. Churchill, V.M.D.

DR. ROBERT R. MARSHAK: As far as I have been able to determine, this disease has been almost entirely neglected, from the research standpoint. Fortunately, there is a close analogy in man. It is called paroxysmal paralytic myoglobinuria and in many respects seems to resemble the tying-up syndrome of horses. Both of these diseases and, also, azoturia, the old classical disease of draft horses, appear to be related to defects in muscle metabolism. I cannot see how one can avoid a consideration of the fundamentals of muscle metabolism and the chemistry of contraction in thinking about these disorders. Thus, it seems appropriate to consider some of the theoretical background, and then some preliminary chemical data on several cases we have studied.

[Slide] In the chemistry of muscle, this is a fundamental equation. The little diagram represents a muscle spindle which, when it is excited by nervous stimuli, will break down ATP (adenosinetriphospate), a high energy compound which provides work energy to the muscle cell. Actually, there is more to it than this.

ATP is not only needed for the work of muscle contraction and for the maintenance of tonus in muscle, but it is also needed to continually renew the biological and chemical organization of the muscle cell against the dissipative forces which metabolism and work bring to bear. Energy, in other words, is needed to maintain the internal environment against electrochemical and concentration gradients which are constantly attacking the cell.

The reason this is a high-energy compound is that its terminal phosphate bond, when broken off, results in the release of energy. These considerations are essential in thinking about muscle metabolism and the myopathies.

ATP breaks down to adenosinediphosphate plus phosphate plus energy. Some of this energy is in the form of heat, and some is free energy which is available to the cell for work. These latter two substances are quite strongly acid.

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Next, I would like to consider what happens between blood and muscle, in the chemistry of muscle. But first, a word about myoglobin. Myoglobin is a respiratory pigment present in muscle cells which, like hemoglobin in the blood, has a great affinity for oxygen. In fact, the affinity of myoglobin for oxygen is much higher. If you had myoglobin on one side of membrane and hemoglobin on the other, the myoglobin would take the oxygen up more quickly and more efficiently. Myoglobin is approximately like the hemoglobin molecule except it has a much smaller size. Its molecular weight is 17,000 and the molecular weight of hemoglobin is 68,000.

This difference in size accounts for the ability of myoglobin to go through the glomerular filter with great ease. In tying up, the coffee colored urine is due to myoglobin. Myoglobin seeps out of the muscle, enters the circulation and then passes into the urine. Myoglobin is important in the aerobic phase of muscle metabolism. It acts as a reservoir and, also, sort of a shuttle for taking oxygen from the blood and bringing it into the muscle cell.

Interestingly enough, when a muscle is at rest, instead of using glucose and other carbohydrate substrates which seem readily available in the blood, the resting cell in most mammals, and I do not know that the horse has been specifically studied, uses lipid substances. These lipids are channeled into the tricarboxylic acid or Krebs cyclevia acetyl-CoA.

After a series of complex chemical reactions, the substrate is burned with the production of carbon dioxide, water and energy. That is the aerobic phase and seems to regenerate ATP.

There is also an anaerobic phase of muscle metabolism. That is represented above this line. The anaerobic phase involves muscle glycogen which is formed from glucose. Glucose enters the cell from the blood and gives rise to muscle glycogen. In the absence of oxygen, when there is energy needed and work must be done, the glycogen will break down through a long series of reactions to pyruvate and then lactate. Lactate quickly passes out of the cell. Quite a bit of energy is produced in the form of ATP, though not as much as in the aerobic cycle. Yet, in a sense, anaerobic metabolism is more important in the working of muscle than aerobic metabolism. Lactic acid, a strong acid like ADP and phosphoric acid interestingly enough, is almost never, under the present concepts, re-utilized by the cell. For example, it could go back to pyruvate and thence be channeled back into the TCA cycle. It does not do this, which is probably a fortunate thing. Instead, it is taken out of the circulation by the liver to form liver glycogen.

This shows you now what happens to a muscle cell at the time you first call upon it do to work. This represents a relaxed cell. This is an actomyosin fibril. In the presence of potassium and magnesium, the cell will contract. Strangely, enough, the

present concept is that relaxation and not contraction of muscle requires most of the ATP. ATP, in the presence of calcium, will permit the fibril to relax again. When this happens, it is the same equation as I showed in the first slide. ADP and phosphate, strong acids, are liberated. There is enough ATP, perhaps, to give you a half second's worth of work. To get more work out of the cell, there is a substance in muscle called creatine, which is a high energy acceptor. When creatine combines with high-energy phosphate, we have a storehouse of energy called phosphocreatine. This can restore the ATP needed by the cell. The ADP (from original ATP) plus the phosphocreatine provide enough energy for about two more seconds work. About 2 percent of the creatine is formed into creatinine, which passes out of the blood and into the urine. Most of the creatine is transported to muscle by red blood cells and is available to form phosphocreatine.

Finally, a tiny bit more energy is available to the resting cell. This is derived from the breakdown of ADP to adenosine-monophosphate.

This is all that is readily available to the cell at the time it first contracts, until it has to call upon its glycogen reserve.

In the absence of oxygen, or when there is oxygen debt, muscle glycogen is broken down to lactic acid for energy. One thing to keep in mind is that a very acid environment is developing in the working cell. Lactic acid is a strong acid, ADP is a strong acid, and phosphoric acid is a strong acid. It is known in normal muscle that the pH can fall to about 5.2 without damage to the cell.

It is thought that during hard work the cell depends upon anaerobic rather than aerobic metabolism because ATP is produced at a faster rate by anaerobic metabolism even though you can get more total ATP in aerobic metabolism. This gives the muscle cell the ability to do hard work, to have a sudden forceful push of energy. This is a very important consideration in muscle metabolism.

When muscle starts to work, the blood supply to muscle increases many, many fold, so that more oxygen and more net ATP is being formed from aerobic metabolism than at rest. However, at work, about 1/200 of the work of the muscle is derived from aerobic metabolism, whereas all the rest is derived from the anaerobic metabolism.

This is to illustrate the sort of thing the body does to pay its oxygen debt. This animal has had a long, steady run resulting in a small debt. This is Hill's classical description of oxygen debt associated with such work.

If the horse is running from here to here, of course, his rate of oxygen uptake will increase. If he is not going too hard, if he is just jogging at a steady pace, he develops this small oxygen debt. He is permitted to develop this debt because he has these

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anaerobic sources of energy to fall back upon. However, whatever you borrow, you must repay. At the end of the run, he has to keep breathing for this length of time (above the base line) in order to repay the oxygen debt he has incurred to bring back the cell to its normal energy status; at rest, he repays his oxygen debt.

This slide illustrates what happens in violent exercise, such as a very short dash. You incur a very large debt which takes a much longer time to repay.

One of the things worthy of consideration is the possibility in horses tying-up, that under certain circumstances they develop such a large oxygen debt that they cannot repay it, and tissue anoxia may be of long enough duration to cause permanent damage.

[Slide] We have some preliminary data on horses with the tying-up syndrome. These next slides show some of the biochemical changes in the blood.

This is a draft horse that had myoglobinuria (azoturia). He died in renal failure.

This is a horse that tied up, a hunter. This is another hunter that tied up. This is the control before exercise and after exercise.

These are enzyme levels in the blood. You are all familiar with the enzyme, transaminase. It is an intracellular enzyme which appears in high levels in the blood in certain conditions. The draft horse with azoturia had an SGOT level of about 2250. The horse that tied up had a level of about 8400. This horse had a lower level. These two controls are about the normal range. It was elevated in the draft horse that had azoturia. In mild tying up it did not vary very much from normal.

In the case of lactic dehydrogenase, there was an enormous level in the draft horse with azoturia. In the tying-up horse it was highest, and in the mild tying-up it was also high. These are normals before and after exercise. What this all indicates is that enzymes which do not normally appear in the blood in such high levels do so in the tying-up syndrome. In other words, it is evidence of muscle necrosis.

Here are some other determinations on these same animals.

Earlier we discussed creatine and creatinine, creatine acting as a storehouse for high energy phosphate. You will notice in the draft horse, and this draft horse was dying at the time this was taken, the level of serum creatine was 5.3 mg. %; in the horse badly tied up 8.1 mg. %.

Creatinine was about normal in all of them, which is not unexpected since the level of creatinine depends on the muscle mass. Not much would be changed by a slight degree of necrosis.

All of these animals had myoglobinuria; all had coffee colored or red urine.

You notice this hunter that tied up so badly had about double the level of urine creatine as compared to the two controls. Creatinine was about the same. These were not 24-hour samples, so they are only of limited value. This indicates, again that serum creatine may be leaking out in excessive amounts, because there is necrosis.

[Slide] These are some other determinations. The only one I would call to your attention is serum lactate. In the horse that tied up badly — (we did not get one for the draft horse) — it was 19.3 mg. % which is about double normal. In this horse even less than normal; in the controls, about the normal level, between 8 mg. % and 10 mg. %.

[Slide] These are some of the values for electrolytes in the serum. The only one I will call attention to is potassium in the azoturia case. All the other constituents are essentially normal, including the total carbon dioxide.

This azoturic draft horse had renal damage and severe muscle necrosis, compared to tying-up horses. These factors may account for the hyperkalemia.

Some people have speculated that hyperkalemia may be the cause of death in some cases of azoturia because of the effect on the heart.

[Slide] This is a section of muscle from a prize fighter with myoglobinuria. We do not have horse muscle slides ready, but the lesion appears to be identical. You see that there is almost complete necrosis with hyalinization, shredding and tearing of the fibers.

[Slide] This is a higher magnification. You can see the edema and other changes in this necrotic muscle.

[Slide] This is an attempt to classify this disease. I prefer to call this group of diseases necrotizing myopathies. There is not basically an inflammatory reaction. Both azoturia and tying-up are exertional myoglobinurias.

The azoturia in the draft horse is much more severe for reasons we do not yet understand, and there is renal damage. In tying-up, it is mild. Renal damage may or may not occur. It is known in man that renal damage does eventually occur as a consequence of a number of mild attacks.

The point of all this, then, is where do we begin to look for some of the answers? I feel that it probably will not come from a study of various constituents in the blood, because about the only thing we have learned from studies of the blood is that there is evidence of necrosis.

Where we must begin to look is in the muscle mass itself, through studies of muscle metabolism.

We are also beginning to study myoglobin. It is possible that in the myoglobinuric diseases of horses there may be a deficient myoglobin or an abnormal myoglobin. This can be studied by

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various modern technics, including electrophoresis. If such proved to be the case, these animals might have a defect in aerobic metabolism and would not be able to maintain their muscle cells normally.

It is a very complex problem, and we are a long way from knowing the answers. We need horses which tie-up regularly and are freely available to interested and qualified investigators.

I hope this discussion has pointed out how dangerous it is to accept therapeutic recommendations at this time. You have to think in such terms practically, but it is really quite risky when there is such a meagre basis for our understanding of the problem.

Thank you. [Applause]

Our next speaker will be Dr. Keown from Washington State.

G. H. KEOWN, D.V.M.: After hearing Dr. Marshak's very able presentation, I think there is not too much left that I can say about the disease. He finished his talk with a proposed classification.

I also think that this disease should be classified. It has been called by quite a number of names by different authors. Blaxter who has done a good deal of study on muscle diseases in the domestic animals, has pointed out the term "myositis" is usually used in connection with bacterial or viral or parasitic diseases, whereas myopathy is related to muscle changes that are not due to these causes. He also points out that there may be dysfunction of the muscle due to neural changes, or neural damage, such as injury to a motor nerve.

In view of this, it would seem that the term "myopathy" would be more suitable or possibly muscle "dystropy" in contrast to "dystrophy" which indicates damage caused by faulty nutrition to the muscle. An example of this is seen in vitamin E deficiency in calves and lambs where a muscular dystrophy develops. A case of muscular dystrophy has been reported in a foal by Jones and Reed.

Personally I do not think that azoturia and tie-up are the same disease. Blaxter also mentioned that interference with muscle, or muscle effects due to totally different etiological factors can produce the same ultimate effects.

So, although similar muscle damage may occur in both, the mechanics of their development is different. No one can deny that there is a very close relationship between azoturia and tie-up. In both, the muscles of the lumbar region and the gluteal region are affected. In both, it is more likely to affect fat animals. In both, recovery may take place in a few hours.

We also see some differences. In azoturia, it is characteristic to find discolored urine due, as Dr. Marshak pointed out, to a loss of myoglobin, the red pigment from the muscle; in tie-up this finding is not usual. Also exercise is considered quite harmful after the development of azoturia. It may result in disaster or

death of the animal. Most veterinarians disapprove of walking a horse that is tied-up but I do not think that it can be denied that they usually will recover it in spite of being walked.

Another difference appears to be that azoturia develops shortly after the animal begins to work. I know there are exceptions but most cases of tie-up appear to develop at the end of the work.

Another difference is that in azoturia the mortality is fairly high whereas in tie-up this is not the case.

The treatment is very similar in both diseases. Some years ago Badame reported that he had had success in treating tied-up horses with an agent that was active in the muscular-neural junction. As the nerve impulse is sent along its fiber is causes the release of acetylcholine at the muscular-neural junction and this causes depolorization of the muscle end-plate and the muscle contracts. The actual process of contraction is associated with complex chemical changes which have been ably reviewed by Dr. Marshak. This is accompanied with the release of heat so that there are actually four changes that take place, electrical, chemical, physical and thermal.

If there is a deficiency in the production of acetylcholine, muscle contraction will not be normal. Or, on the other hand, if there is an axcess of acetylcholine muscle contraction will not be normal.

The treatment that Dr. Badame suggested was the use of physostigmine which augments the action of acetylcholine. He also suggested the use of atropine, because this blocks out the effects of physostigmine on the digestive tract, so that you get a selective action on the muscles.

I would like to point out that this can only take place when a deficiency is present. The beneficial effect of physostigmine can only occur in the case where we have a deficiency of acetylcholine at the end-plate, because an excess will have detrimental effects.

The succinylcholine, which is so commonly used today, actually creates an excess of acetylcholine at the end-plate, causing a dysfunction of the muscle or loss of function.

I know that time is running on so I am going to cut this short and give my theory, and it is entirely a theory, and I will have to make apologies to the physiologists and pathologists in so doing, but at least it will be a theory that someone can dispute or agree with. The theory is that in the case of tie-up, the condition is brought on by a spasm of the blood vessels of the skeletal muscles. There is a contraction of the arterioles. The animal is exercised and less blood reaches the muscles at a time when more is needed. This interferes with function at the end-plate. In addition to this but secondarily, lactic acid accumulates within the cell. The chemical changes within the cell appear as a later development.

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In the case of azoturia I suggest that it is primarily a chemical disturbance originating within the cell. It has been stated that lactic acid is extremely hydrophilic and when it accumulates in the muscle cells in excessive amounts, swelling and hyaline degeneration results. In contrast to tie-up early necrosis is the characteristic of azoturia as evidenced by the release of myoglobin. The secondary effects on the heart muscle and kidney and high mortality in severe azoturia is the result of the blood being laden with toxic products from the damaged muscle.

Another disease that has been linked with discussions of tie-up is traumatic myositis or race horse disease as described in Huyra and Marek. This appears to be quite different from tie-up or azoturia. It is stated that this condition is a strain of the muscles where there is actual tearing of the muscle fibers.

In view of this, I suggest that there are three entities involved in these muscular dystropies, namely, azoturia, tie-up and traumatic myositis.

Our next speaker will be Dr. Adams from Colorado State.

DR. O. R. ADAMS: I thought maybe the best thing I could do would be to try to summarize. I am going to differ a little bit, perhaps, but that is why we are here, to try to get across all the ideas we can.

First of all, I think that azoturia and tying-up are related. I think it is a matter of degree. I believe that when a horse has been rested and has accumulated a considerable amount of glycogen in the muscle, it is broken down very rapidly when the horse is worked. Lactic acid is formed very quickly under these circumstances. This is what happens in azoturia and considerable muscle necrosis results.

In tying-up, the animal is in better condition and builds up the lactic acid level more slowly. I do not think there is any necrosis of muscle in tying-up. In typical, reasonably mild tying-up, it recurs many times. If there were much necrosis of muscle, the muscle would be mibrotic and nonfunctional.

[Blackboard] Very briefly, here is what happens. There are two sources of energy, as has already been indicated. There is anaerobic energy and aerobic energy. Our primary interest is anaerobic energy. Let us say that through glycolysis, lactic acid results, after the anaerobic oxidation. If oxygen is present in a sufficient quantity, lactic acid will quickly be converted to pyruvic acid. Pyruvic acid is broken down through the Krebs cycle and ends up as carbon dioxide and water.

What you as practitioners are interested in, is that anaerobic oxidation is the source of energy the animal gets in a race. He has to use a considerable amount of anaerobic oxidation, and ends up with lactic acid. We know that experimentally following repeated stimulus of muscle, lactic acid production causes rigor or spasm. When the lactic acid is removed, the muscle returns to normal.

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The lactic acid is removed by way of the blood stream and is converted in the liver to glycogen and glucose. It is also removed by oxidation, if enough oxygen is present.

So, how should we treat this? I say that we should treat it: 1) by alkalinization of the blood stream. It is a known phenomenon that the blood pH and the pH of the affected muscle do not necessarily reach the same levels. In other words, the blood will show a more nearly neutral pH than will actually be present in the affected muscle. It is not known why this happens. So, because we think lactic acid is present, we think we should alkalinize. We can do that with sodium bicarbonate intravenously if the case is serious.

2) Thiamine may be given to aid in breakdown of lactic acid. Thiamine aids in decarboxylation of pyruvic acid. It is essential by acting as thiamine pyrophosphatase in breaking down lactic acid. Oxygen would help, but the only way it could be given effectively would be to give it under pressure; since this is impractical it is not used.

I would like to state something about what I really think is the cause of tying-up. I think that most horses are not conditioned properly. Of course I think there are individual variations. I know this is very true in athletes. An athlete must train if he is going to do well in a sport and not get a charleyhorse or cramps.

Most of the race horses are not trained. You cannot train a race horse in one hour, put him in a box stall for twenty-three hours and expect him to race. That happens in a lot of cases: Also, there is a lot of difference in individual people or individual horses. One of you may be able to train a shorter period of time for a sport and he in a better condition than someone who has trained in an identical way.

If a race horse is not properly trained, this will lead to a quicker accumulation of lactic acid. If properly trained the horse can overcome this deficit in oxygen. [Applause]

Our next speaker will be Dr. Lundvall from Iowa State.

DR. R. S. LUNDVALL: I think the subject has been covered quite well, and the time is getting late. I have a feeling that we are in the same boat as the M.D.'s when we are talking about this condition, that we are lumping together several conditions, rather than one.

I do not believe, personally, and I am probably wrong, I will admit it, there is any muscle damage in tying up as in azoturia, and there are several other things that we probably are lumping together all in one. I think quite a little research must be done to straighten these out.

The treatment, as indicated before, must be varied to the particular condition. For example, in azoturia, there is muscle necrosis. As I stated before, I do not believe there is any muscle necrosis in tying up.

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Then the race horse disease which lasts for a period of several weeks, certainly this must be a different entity. This condition lasts for a long time, and still there is no muscle atrophy as we see in azoturia.

Mention was made of the human condition, myoglobuinuria, paroxysmal myoglobuinuria. If you search the literature carefully, and talk to the M.D.'s about this, you will find that they are not only confused, they are more confused than we are. The definition that we find now is "unknown etiology affecting skeletal muscle, with liberation of myoglobin." There are other diseases that they have classified in man, thiamine deficiency, which probably accounts for a good share of some of our cases of so-called tying up.

There are other things that can happen in man, and there is no reason why they cannot happen in animal. The so-called crush syndrome, actual injury to the muscle, will liberate myglobin and produce some of these symptoms.

As I said before, I think it has been covered very thoroughly, and the thing to do is to determine whether you are dealing with azoturia or the typical tying-up that we think we see in some of these animals. There is one other possibility; a myoglobinuria that is a family characteristic in humans. It is an inheritable factor. In other words, instead of one simple thing, we may be dealing with four or five.

The treatment necessarily must wait until these have been differentiated. Thank you. [Applause]

MODERATOR BRENNAN: Thank you, Dr. Lundvall.

Our next speaker will be Dr. Churchill, practitioner from Centerville, Maryland.

DR. E. A. CHURCHILL: I think we have covered the basic possibilities in this disease, and all I am going to do is to open the door and leave it open on, perhaps, some of the clinical considerations and some ideas that come to my mind. Anything I say here I may have no basis for other than my own observation. I do not expect you to agree with me. This is just some of my thinking.

First of all, going back to the time when we start training these horses, I have often thought we get these colts excessively fat before we sell them as yearlings. Oftentimes we refer to them as hothouse colts. We put them in training, and go from there.

It would seem to me that, when you are dealing with an animal that has an articial amount of fat on its body, we are much more apt, perhaps, to produce some damage in the early training that was not, perhaps, apparent at that time. I cannot help but feel that might have some bearing on the ultimate factor as to whether this disease will appear later in that animal's life.

In my mind, as I see one of these cases at the race track, I do not always consider it a horse that is exactly tied up. I do

think that the term "tied up horse" is related to the azoturia. I do think it is a matter of degree.

However, when somebody comes around and wants you to run right over as they have a horse tied up, many times you will find this horse actually does not have any muscular ailment at all. Many horses will walk, walk like they are tied up, right after they come off a race track, as a result of being footsore and lame, and because he has put undue exertion on muscles that are not used to being exerted. He is just muscle weary, which, perhaps, would be a better term, in an attempt to compensate for some lameness, or other defect in the horse.

Another thing I notice is that the true tie-up cases, that is those that do occasionally throw off myoglobin in the urine, are usually horses that are good feeders and good doers. That is one classification.

On the other hand, we have horses that apparently tie up occasionally, and most frequently in the same stables, that do not look like the kind of horse that ought to tie up. These are horses trained down to a frazzle.

In these thin horses or highly trained horses, whatever you want to call them, I think they are not exactly tied up as much as they are muscle-weary. If any of you have ever participated in sports that required a great deal of exertion, you know that, following great exertion, you actually go numb in the muscles, or some of the muscles that you are using. I think a horse will do the same thing.

I think, depending on the group of muscles involved, you can produce all sorts of gaits, just due to weariness on the part of the animal, and complete exertion.

Then we get down to the horses that would, in my classification, fall into the typical tied-up group. No one here this morning, so far, has offered to me any explanation as to why about 95 out of every 100 horses I treat for being tied up are mares. I do not know whether that is the condition all over the world, or whether that is just a condition where I happen to practice, but this seems to be quite a factor.

The way I have explained it to myself is that I feel that the mare is more of a temperamental and highly nervous animal than is the gelding or the colt. I think that somewhere in the equation of this disease there has to be some influence exerted by the nervous system. Perhaps this would have to do with hormones. But going over the basic endocrinology, as far as I can go over it, which is not really a good analysis, I cannot figure how any fe-female hormones would alter the basic picture of this disease. Therefore, I blame it on the nervous system.

I agree with Dr. Adams that these young colts are possibly under-trained and, as a result of being under-trained and carry-

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ing a lot of weight, it may be a factor in those horses being tied up. I do not agree with him, though, on the basis of older horses.

I find most of these cases in horses or in stables that are trained regularly. I think that, actually, regular training is a predisposing factor to this disease, in a way. It would appear to me that we are trying to condition this horse to be able to race, by training him, and by training him we are building up his capacity to store glycogen in the muscles, and increase the amounts of glycogen in the muscle so he can use them, when he races. I think this is an important part of the training program.

Of course, when you get the horse used to storing an excess amount of glycogen as a result of this training, and then do not train him, or lay him off for any reason, then it is easy to see how he would have an excess amount of glycogen break down upon exercise two or three days later.

I have a lot of trouble with getting horses from one race track to another, on this basis. I have developed a program—I do not know whether it is good or bad; it works sometimes, so that it is better than nothing—of starving these horses, restricting the grain prior to shipping, and not graining these horses again until after they go back to exercise. Usually these are the horses that are fat enough to stand this sort of treatment; I mean, they are not going to starve to death in the meantime. They are the horses that are good eaters. The only prerequisite to this sort of program is to ship early enough so the horse has something to run on after he gets there, particularly in the case of a stake horse.

As you probably can conclude from the previous discussions, the treatment is strictly of choice. Everyone has his own program, and I do not think anyone treats these things the same way day after day, or at least that has been my observation.

Theoretically, a lot of these treatments that we use do not have any foundation. The administration of thiamine, of course, is a very popular treatment and this, of course, has some foundation. I think most of us use thiamine in the treatment of these horses. Calcium gluconate is also another substance used in the treatment. Here we think possibly we are supplying calcium that may be depleted, and it is necessary for normal muscle metabolism. I think, however, that our calcium therapy is more referable to the type of case that I described earlier, that is not the fat horse, but the thin horse that is muscle-weary. He responds well to that type of therapy.

I think it is quite apparent we have toxemia where the tying-up is severe. Therefore, it would be in line, I think, to attempt to relieve the toxicity of the horse. This could be done by the administration of fluids, or by the administration of so-called kidney medicines. But here, again, the administration of diuretics should be carefully considered, because those diuretics that act directly

on the kidney tissue, should be avoided in a case where you have a secondary nephritis.

I have used cortisone a great deal in these cases, or first cousins to cortisone, and with some success. If I have a case that does not respond to the ordinary therapy, I will frequently have them hold the horse until I get to the track in the morning, administer cortisone intravenously, and then have them wait about an hour before they take the horse out of the barn. This frequently will get you off on the right track.

In line with blaming the nervous system for part of the syndrome, I found, particularly in those horses that "grab on the iron" as soon as they hit the race track, a very small dose of Sparine, perhaps 150 mg. will be enough to calm them, and make them easier to control. I think it prevents, to some degree, the production of the symptoms.

I heard someone mention they use Sparine alone in these cases. Perhaps that would work, but I never use just Sparine alone in treating these horses.

I think that is all I have. [Applause]

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INHALATION ANESTHESIA

CHARLES ALLEN, V.M.D.

WILLIAM O. REED, D.V.M.

I am simply reading this paper for Dr. Allen. He has much more experience in this than I will ever have. In fact, there is an old statement made by surgeons which states as follows: "The best friend a surgeon can have is the anesthesiologist." That is true in this case. Dr. Allen is a good one. The paper reads as follows:

"A more appropriate title to this subject would be 'The Use of Fluothane as an Anesthetic Agent in Horses', as this paper will evaluate the uses and advantages of Fluothane and the apparatus used.

"One of the obstacles confronting the veterinarian concerned in large animal surgery has been the inability to have the proper anesthesia in regard to: 1) rapid induction to the surgical stage, with as little excitement as possible; 2) the ability to change rapidly and safely to and from the various planes of anesthesia; and 3) the all-important recovery stage following surgery.

"The recovery stage has presented a great many problems and, as has been mentioned before, is of great importance to the patient's welfare; prolonged restraint, injuries, et cetera, and the number of attendants required to assist the animal while recovering from anesthesia, and the dangers they are exposed to by a wildly thrashing animal.

"Various methods and technics have been developed and tried in order to obtain the desired end results, but up until now we have not found the most desirable agent or apparatus. Various gases, such as ether, chloroform, or combinations of gases, have been used, but these units, or modifications of these units, have been discarded as unsatisfactory in producing the desired results.

"This paper and the accompanying film will demonstrate a satisfactory anesthetic agent and unit. We will not attempt to discuss the physiology of Fluothane, or the various types of apparatus used, but the unit which has been used by us.

"The unit consists of four individual vaporizers which, in turn, connect to a common duct. The oxygen passes from the cylinder and regulator, which we have set at an arbitrary pressure of 50 p.s.i. through a common inlet where each individual vaporizer is supplied. Four vaporizers are used, and each is controlled individually in regard to 1) the litters of oxygen per minute and 2) the percentage of Fluothane produced.

"The liters of oxygen produced are controlled by a gauge at the top of each vaporizer and are graded from 0 to 20 liters. The percentage of the Fluothane gas is regulated by a circular dial at

the top of each vaporizer and is calibrated from 0 to 4.4, the maximum setting.

"The maximum output is obtained with 8 liters of oxygen per minute and a dial setting of 4.4 which will produce a percentage of 4.8, that is from each individual unit.

"These vaporizers are controlled one at a time, and the percentage may be raised or lowered, as the operator deems necessary. The outlets of each vaporizer connect to a common duct by way of one-inch rubber tubing which, in turn, leads to the soda-lime canister for the CO_2 absorption; while the terminal portion of the canister has the 60 liter rebreathing bag. The other end is fitted with the equine mask.

"This unit is easily operated by one man, and with a minimum amount of mechanical intricacies.

"The anesthetic agent which has been used is Fluothane, a nonvolatile liquid, and which has proved successful in obtaining our desired results. As compared to the other volatile gases and barbiturates, Fluothane has the following advantages: 1) It is a nonvolatile gas; 2) produces rapid surgical anethesia; 3) the depths of anesthesia can be easily controlled; 4) rapid recovery time; and 5) the all-important safety factor.

"The use of Fluothane as a large animal anesthetic will be shown. Approximately 60 cc. of Fluothane are put in each vaporizer, and the dial settings are placed to the maximum output percentage of 4.8; these are brought into use individually, as needed.

"The subject shown in the film is a yearling colt, and the surgery to be performed is a fractured splint bone. The patient was given no medication prior to surgery, even though tranquilizing drugs have been used with success on apprehensive animals.

"The initial drug demonstrated here is succinylcholine chloride, 50 mg. intravenously, and this is used to produce a short lasting anesthetic, sufficient enough for the application of the mark. Once the face mask is applied, all four of the vaporizers are brought into maximum capacity of 4.8 per cent. These may be regulated once the proper plane of anesthesia has been obtained.

"Effective anesthesia may be expected in from three to ten minutes, and with little resistance on the part of the animal. Once the proper plane has been obtained, the degree of anesthesia can be readily and effectively controlled, either by increasing or decreasing the per cent of Fluothane and the flow of oxygen. During surgical anesthesia, the respirations may vary from 6-10 per minute and the pulse rate between 30 and 50 per minute. Complete relaxation is obtained for all types of surgery. Another advantage to the surgeon is that there is less capillary oozing at the site of surgery because of vasoconstrictor action of the anesthetic.

"As the surgery nears completion, it is possible to reduce the flow of Fluothane vapor to lighten the plane of anesthesia. This

may be accomplished by completely shutting off one or more of the vaporizers and then administering pure oxygen to the animal.

"An endotracheal catheter, with an inflatable tube and an inflatable cuff may also be easily attached to the tube for administering the anesthesia, and probably ease the control of anesthesia.

"The unit should not be shut off completely before surgery is completed because the patient may come out of the surgical stage within three minutes. Therefore, it is necessary to keep the animal on a light concentration of the gas. When the surgery has been completed and the animal is returned to the floor, there is no restraint or postsurgical medication used; that is, tranquilizers or sedatives. Within ten to fifteen minutes, the animal is able to regain its feet with little or no loss of equilibrium, and with virtually no manual assistance.

"The use of Fluothane anesthesia allows various types of surgery to be done that had not been attempted before, because of the difficulty in controlling the anesthesia. This gas has proved very satisfactory from the standpoint of rapidity of producing surgical anesthesia, safety, the ease of controlling the phases of anesthesia, the rapid recovery stage, with a minimum of excitement.

"Experience with this unit and the anesthesia has been very gratifying in regard to the primary needs of a suitable anesthetic and anesthetic unit."[4]

[Motion picture] This is the animal to be brought on top of the table. That is the Insulite. It is being given succinylcholine here by Dr. Allen.

The unit is being put on here, as you can see, with face mask and the four units as you can see right here, and the soda lime canister on the other end, and this is the rebreathing bag. These are the four individual units, and they come into the common duct at this point here. It is inhaled into the face mask.

The important part, apparently, about Fluothane is being able to control the concentration of the Fluothane. It is a rather potent preparation, and in human medicine they want to control it within one-tenth of one per cent.

He is under just about as quickly as this, too. This, incidentally, is a home made movie, made a week ago yesterday, and is just routine procedure, without any special preparation or anything at all. This is actually just about the way it works. These are micrometer adjustable devices. This is the flow meter. That adjusts the oxygen intake into each unit.

One thing kind of scary, if you notice the rebreathing bag, it does not move too often during this stage. You look up and think he is not breathing, but he is. The respiration slows down to 2 to 5 per minute.

DR. PROCTOR: Is that gas anesthesia or succinylcholine?

DR. REED: No, that is typical of Fluothane. Fluothane is

typically very slow in respiration. Once you take it off, you want to get him ready. Here he made one preliminary attempt to get up. He does not make it this time.

They sit up like a dog and, the next thing you know, they are up. There he is. He doesn't thrash too much. You do not see them struggling or swimming or in any way getting upset. They will stand there acting, I imagine, like a dummy.

He came right on out just about this way. You will see him stand over at the side as though nothing had been done with him. They do not act like they are going to fall down, or act incoordinated. They seem to be bright, as though nothing had been done with them.

Do you have any questions?

DR. PROCTOR: How much?

DR. REED: I think they figured they used on this horse about 28 cc.

DR. PROCTOR: I mean dollars. [Laughter]

DR. REED: You mean the equipment?

DR. PROCTOR: The anesthesia machine.

DR. REED: I don't know. Each one of these is an individual human thing. I do not know what they cost. We rigged them together like a Rube Goldberg device. I don't have any idea what they cost.

DR. PROCTOR: This is professional oxygen?

DR. REED: That is right, professional oxygen. I thought your question was the amount used. It might be well to answer that. I think it was, roughly, 28 cc. I think Fort Dodge has a proprietary name on theirs, they call it Halsan.

Any other questions?

QUESTION: Do you have any trouble with the succinylcholine?

DR. REED: I have a great deal of confidence in it. I have used it on a real mean yearling; used it twice within the period of ten minutes time. I use it on horses insured up to half a million dollars. I do not have any apprehension about it. I don't worry about the possibility of respiratory embarrassment. We make no special provision, with the exception in the operating room we have artificial respiration. There we have it available.

DR. PROCTOR: What is your usual dose?

DR. REED: We use the Burroughs Wellcome preparation. It is usually about 2.4 cc. There are, I think, 20 milligrams to the cubic centimeter,—use 50 mgm.

QUESTION: You gave that particular animal 1cc.?

DR. REED: No, 2.2 cc.

QUESTION: Give them all succinylcholine first?

DR. REED: Yes. One reason is it adapts well to our table. The table, you see, is a hydraulic table. I do not think you have to do it, but it does adapt to our procedure, by following in that

manner. It is easier for us. I think with other procedures, it would be quite adequate. For us it seems to work out all right.

DR. JOHN D. GADD [Cockeysville, Md.]: How about an operation of an hour or hour and one-half?

DR. REED: We had one on for two hours. We had the old ether equipment; we did not have it calibrated. We dumped in some Fluothane. This was more or less of an experimental case. We had this horse out for two and one-half hours. We used all of the Fluothane. We must have used a massive dose. There was no trouble at all. They figured he was up sixteen minutes after we took the mask off of him. If you used an endotracheal catheter—we simply did not have time to rig it up here—I think you could cut your dosage down more.

There is a trick to using the endotracheal catheter. You fellows who pass the stomach tube, the tube can easily go into the trachea. If you extend the head upward, it will slip into the trachea. With the succinylcholine, it has been easy for us, just playing with it.

DR. PROCTOR: You got this horse under anesthesia; you can control the depth of anesthesia.

DR. REED: You can control it to the point that, if you want to, he can move a little bit, or you can control it to the point where he won't move a muscle.

DR. PROCTOR: How about the rapidity of respiration? Is that any indication of the depth?

DR. REED: That is. When they start increasing their respiration, and pupillary reflex is a big factor—that is, perhaps, the biggest factor of all. They will tell you, with that, where you stand.

DR. H. W. JOHNSON [Littleton, Colo.]: Have you used Sparine, then Sucostrin, with atropine, under any conditions?

DR. REED: Yes, we have used it when we used ether, but we do not use it with this, because this has the effect of lowering blood pressure, and I think Sparine would be a little contraindicated with that. I think that might be a possible disadvantage. We did use Sparine routinely, as a preanesthetic tranquilizer, and atropine, also, when we used ether.

DR. JOHNSON: Did you use that combination?

DR. REED: Yes, we did.

DR. JOHNSON: Any bad results?

DR. REED: We never have. Have you? I mean, I am asking.

DR. JOHNSON: The reason I asked was I tried it just once and immediately when I got the effects of Sucostrin, the horse went straight up in the air and when he hit the ground he was dead. I was wondering about that particular combination.

DR. REED: This might be all wrong physiologically but we have used the atropine once we have started, we have gotten it down, not initially. We gave the Sparine 20 or 25 minutes ahead

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of time; then used the succinylcholine chlorile. Then started them out and gave them atropine, too.

I had a bladder stone we took out. We had a human anesthesiologist there that day, and this was a lady. She was taking blood pressures, sort of experimentally on this horse. She had that at one stage when I was in the bladder she noticed the blood pressure going down markedly. I said, "What do we do?"

She said, "We do one of two things: stop and wait or administer atropine immediately."

They gave the horse some atropine and everything was all right in about five minutes.

QUESTION: Can you use succinylcholine intravenously or just with gas?

DR. REED: Yes, you can. Every once in a while at the race track we get in a spot out in the field where we want to knock a horse down; we don't want to completely knock him out. We will use a combination of chloralnitrosulfate and succinylcholine preoperatively first. We had one that kind of scared us. We had to jump on his chest a few times to initiate rebreathing. I think you can use it, providing you do not use a massive dose immediately. Let the horse start to come back a little bit. I think it has a wide margin of safety. [Applause.]

X-RAY TECHNICS AND DIAGNOSIS

Moderator: FRANK J. MILNE, O.V.C.

Panel: D. D. DELAHANTY, D.V.M.

D. L. PROCTOR, D.V.M.

BERNARD F. BRENNAN, V.M.D.

W. F. RILEY, JR., D.V.M.

This panel all came about because I took Dr. Woodcock up on his suggestion that we might give suggestions to the Program Committee. Before I knew where I was, here I am! Next time I will keep my mouth shut.

The big difficulty, as you can see, is the showing of the films. We had no idea how many people would bring x-rays, and we had no idea what type of x-rays we would have, what the response would be. I think everyone will agree that the best place to discuss x-rays films is in front of a viewer, where you have a magnifying glass, if necessary, and extra strong lights. We have none of those here today. So, I am going to ask our panel to come and sit fairly near the front here, and I am going to show on the opaque projector some films that have been handed to me over the last couple of days. The response has been terrfiic; in fact, so terrific, you will probably see everybody's x-ray plates but your own.

Anyhow, case histories are at a minimum, and that is a little bit hard on the panel because whatever anybody may say, I think history is of value.

I get a lot of plates sent in by practitioners, and I get stuck, too. That is why I am here with some of my plates to push to the panel and open the general discussion.

May I quickly ask one of two things: stick an identification mark on your plates, and if you appreciate the fact that photography and radiography are closely allied subjects, I am quite sure you would not let Junior play with all the negatives you so jealously store away, yet finger prints and x-rays don't go too well together, but you would think they did, sometimes.

I will be able to show you a case today with regard to the foot. Might I make a plea that the part be cleaned out thoroughly beforehand. We literally have had rocks wedged into the frog, and underneath, because the fellow did not take time to clean it. Those are wonderful foreign bodies. We took the foreign bodies out, and everybody was happy, but that should not have been necessary.

X-ray is an aid to diagnosis. Many lesions may be present. We have to find out if they are significant. That is part of the fun and bafflement of x-ray diagnosis.

Would the members of the panel please come down toward the

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front? We have with us Dr. Delahanty, Dr. Proctor, Dr. Brennan and Dr. Riley.

In advance, I am going to apologize for those fellows. Up till now we have not yet been able to get together. This is the first time we have all been together. Everybody is going to play it by ear, and they are dead cold.

This first plate was submitted by a practitioner. It is actually an excellent radiograph, but the facilities here do not show it up too well.

Dr. Delahanty, this is the one we did have a look at last night. Frankly, I do not think anybody would blame you if you did not make a diagnosis on this one. Can you remember it? There is very little history of this horse. He did show swelling, and was actuely lame—just a yearling—after workout. X-rays were taken four or five days later. There was an area of pain on the medial aspect, below the carpus. I do not think it is going to be too good.

DR. DELAHANTY: I think so, Frank. This is Dr. Copelan's case. There is a discrete point of tenderness in the leg, right below the carpus. Perhaps some of you can see a slight difference in density from here to here, with the suggestion of a line right here. I think this may well represent an area of localized aseptic necrosis. It may be traumatic in nature. It could also be thrombosis of a vessel in the area. I kind of think that is stretching a point. Traumatic lesion or even maybe fracture, with a form of sequestration coming on. That is just my guess, that is all. I wonder what the rest of the panel has to say on it?

MODERATOR MILNE: Dr. Proctor, would you like to comment on this one?

DR. PROCTOR: I would concur with Dr. Delahanty. I have had a similar lesion that turned out to be a subperiosteal homatoma. If you get them early and aspirate them, you can do some good. Otherwise you get sort of a non-septic necrosis.

MODERATOR MILNE: This is an x-ray submitted by one of our members, and I am sorry I haven't got too much in the way of history. Somebody did write something on the envelope, but I guess he writes like I do, and that is not very helpful. As far as I can gather, this is an 18-year-old, five-gaited mare, and it was limping on the toe, right hind leg.

Dr. Brennan, would you care to comment on this plate? That is all the history I have.

DR. BRENNAN: Really, in looking at this, it looks like the area of the navicular bone may be involved. That black area certainly looks like there might be some extra calcification.

MODERATOR MILNE: I have another one of the same horses, I will change it over and let you see that.

DR. BRENNAN: There is overriding. I certainly feel there is some joint change between the third and second phalanx, prob-

ably some degenerative changes taking place there. I will pass this.

MODERATOR MILNE: Would you like to have a look at the close-up plate? That might help. There is an awful lot of detail lost on those, when you enlarge them on the screen.

DR. BRENNAN: There is some calcification up high on the second phalanx, also the os pedis there is pointing down, which would indicate probably we have some pathological changes in the soft issue. If this were on the front leg, you would anticipate that probably we had a founder case here, a case of laminitis.

I have interposed a couple of suggestions. I will just pass this on to Dr. Riley. He may have some additional comments.

MODERATOR MILNE: Dr. Riley, would you like to look at the original and see if there is anything to comment on?

DR. RILEY: On the original radiograph, you can see distinctly that calcification up at the proximal extremity of the second phalanx, and there is very definitely a dropping away, it looks like on the film, of the anterior surface of the third phalanx. There is peculiar mottling on the anterior surface of the wall, which may be dirt. I have not usually seen that in an x-ray. I concur with Dr. Brennan.

MODERATOR MILNE: The next two plates I am going to show just to prove the point that one should never depend on a single view, that at least two should be taken.

For instance, when we put this one on the screen, you do not see an awful lot of pathology. You see things that are wrong. There is subluxation and, actually, there is quite a spur that does not show too well.

When we look at the A.P. would you go for ringbone? I think anybody would say there is a ringbone in this case.

On the next slide I have no history. I cannot help you. There is nothing on the envelope at all, and I do not know if this is going to show up too well. I think if I remember rightly, in conversation, this horse was very lame, obviously a foot lameness, according to the clinician. It was blocked—I do not know whether it was a high or low block—and the horse went sound. I rather fancy it was bilateral high, which would indicate trouble was in the foot.

I believe the clinician's diagnosis was possible pedalostitis. Is there enough, Dr. Delahanty, to start off with that? I have another plate. I do not know which is the better of the two.

DR. DELAHANTY: Is it possible there is a little fracture line here? I would like to see a little more dense film. I grant you the horse shows relatively acute pedalositis, with a sclerotic type of change. What it looks like way in the back of the room, I would not be prepared to say. I cannot see too much up above that, but the whole film is mottled. I do not like to criticize technic, but one of the basic ways to find out whether film is properly exposed and developed is to put your finger behind. If you can see

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the shadow of your finger on the illuminator, your exposure has been inadequate or your development procedure has been inadequate.

MODERATOR MILNE: Dr. Riley!

DR. RILEY: I may be misreading it but it looks to me like there is some change on the compact bony tissue. It might be a fracture of the second phalanx. What leads me to think this, there is a little fissuring right here. That isn't an artifact in the film, I don't believe.

DR. DELAHANTY: I am going to argue a little bit. I think it is an artifact. The only reason I say that is an artifact, this line carries down off laterally over in here. I just wonder whether this horse hasn't a pretty high heel with a shrunken frog, and this is a little filth in the crevice. That is just a question I raise.

DR. RILEY: Difference in density?

DR. DELAHANTY: The density is there. If this were present in two views, you would take it as a good suspect for fracture, probably, but only in one, you kind of raise a question in your mind.

DR. ROBERT H. BAKER [Montclair, Calif.]: Is the lower portion of the navicular bone a little roughened?

MODERATOR MILNE: The question is, is the lower edge of the navicular bone roughened, as the speaker seems to think it is from where he is sitting? Dr. Proctor, you were looking at the slide. Would you care to comment on that at all?

DR. PROCTOR: I would think it isn't diagnostic.

I believe on the original here, on this picture and also the other one, it shows there is a fracture of this wing of the os pedis.

DR. DELAHANTY: Here is another point of order. You move the x-ray around and the fracture moves around. You have a dirty screen behind you. Move your x-ray films on the illuminator and you can eliminate a hell of a lot of fractures in that way. [Laughter]

MODERATOR MILNE: You can see, boys, we are really getting warmed up. [Laughter]

I put this one on because I think the pathology is obvious to everybody, but I would like a comment on the possible etiology of the cause of that pathology.

Dr. Brennan, I think you are next. Would you care to comment on this? Just speak what is on your mind.

DR. BRENNAN: This may be a slide that I submitted. Maybe history will help.

This animal was presented to me more or less by chance. The animal was racing sound. I was doing work for this individual. I was amazed at the size of the leg. We had to x-ray another horse in the barn, so I decided to x-ray this animal also.

The history that came with the horse was that when it was a two-year-old they felt that it was lame in its ankle, and a blister

was applied. From the time the blister was applied, the leg never went down. Certainly, we have extensive exostosis all around this pastern bone, the first phalanx. No one was any more amazed than I when I saw the results of this x-ray, and to know the horse was at the race track and was racing.

This horse, prior to my coming in contact with the animal, had been raced in brace bandages and it went sore. As soon as they removed the brace bandages, the horse became sound and stayed sound. I think, other than those statements, this plate was just more or less to show some of the extensive pathology that can exist, and yet we still have a horse racing.

MODERATOR MILNE: I wonder if I could throw in a question. One of the plates submitted is of fracture of the os suffraginis. This is a condition which we saw a lot of in Britain, on soft tracks, too, yet, as you know, os pedis fracture is never encountered over there. Perhaps the opposite holds good over here. I think in our college records for the last six years, we have had only three cases. In there any possibility this might have been fracture?

DR. BRENNAN: I would certainly think at the time the horse was lame he could have had a fine line fracture of the first phalanx, and they were just unaware of it and blistered the animal. When healing took place, this was the end result. In other words, this x-ray is I believe three years after the original problem.

This is a stifle, and I hope it shows. Dr. Delahanty, I am going to ask you to come up and look at the plate here because I think you will see the lesion O.K. Maybe you will be able to explain it to the audience. We have no history at all, at least there is no history available in this case, to my knowledge.

DR. DELAHANTY: It looks to me like there is a mass of sequestrated bone on the condyle. I wonder whether this might not be what we sometimes call osteochondritis dessicans. There are some irregularities up here, but this big mass in here gives me the suggestion that it may well be a free body, not free in the sense of a joint, but it is there and not attached to the femur or tibial crest, yet we cannot come down here far enough. Certainly it is very smooth and even. Even up in here we see a little irregularity that might be suspicious. I believe that is the big spot there.

MODERATOR MILNE: Dr. Proctor, can you help us on that one at all?

DR. PROCTOR: I feel in the area of the meniscus of the femoro-tibial articulation, there is definite roughness and some abnormality there.

MODERATOR MILNE: Thank you. It is when we come down to those plates that we do not take every day that we get kind of fouled up.

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Dr. Proctor, we can see the pathology on the next one. I would like your opinion, your type of approach to treatment of this and prognosis.

DR. PROCTOR: It shows very, very plainly that this is a fracture of the volar surface or plantar surface of the proximal end of the long pastern. This is a fairly common fracture of the hind leg of standard breds. I do see it in some thoroughbreds. This piece of bone pops off. The surgical removal is uneventful. A filly that was entered in one heat in the Hambletonian had this thing surgically removed. She is now in Canada and races uneventfully.

DR. BRENNAN: I might just comment that everything Dr. Proctor said is correct. That is another one of the radiographs I submitted, and that is just the way it was handled. This was a back leg.

DR. PROCTOR: I feel one of the factors that makes surgery absolutely essential in a case like this is that this is a relatively new fracture. The segment is clean, sharp, clear, distinct. Of course, this is crystal gazing, but I would say if that is not removed in a period of six months, you are going to have enough periarticular arthritis caused by nature's attempt to encompass that segment which had no blood supply of its own, that it will be almost like proud flesh. This resulting calcium, which is nature's attempt to alleviate this condition, is far more serious than the piece of bone. You remove the sequester with the thought in mind of keeping nature from trying to encompass it in healing it.

MODERATOR MILNE: We will go to the next one.

I have a little bit of history. It is a hunting horse, ten years old; it was injured in hunting. Severe pain in the hock on digital pressure and, also, while the horse was in motion, so much so the veterinarian thought they were going to find a fracture or something in here.

The plate is O.K. Dr. Proctor!

DR. PROCTOR: With Delahanty looking over my shoulder, I am a little hesitant to speak out until I have really looked this thing over. [Laughter] To me there is definite lipping at the tarsal—MT3 articulation and, also, above here. That, in itself, would be enough to cause some aberration in gait. It does not look like an acute thing to me. This area in here is a little rough.

MODERATOR FILNE: The question that the veterinarian on the case wanted to ask, was there any significance to the line up here which looks as if it is an epiphyseal line in a horse ten years ago, or is that a projection factor?

DR. PROCTOR: There should not be an epiphyseal line in a horse of that age. This could very well be erosion or breaking away either of the articular surface of the distal end of the tibia or could be part of the trochlea of the tibiotarsal.

MODERATOR MILNE: Dr. Riley, will you please speak on this one?

DR. RILEY: Looking up at the film here I did not see as much pathology as you can see down here on the radiograph itself. I will agree with Dr. Proctor, there is roughness. I do not know what it is, whether on tibia or tibiotarsal bone. There are some lines running down here which could be in the tibiotarsal. I would reserve opinion on that.

MODERATOR MILNE: Dr. Brennan! We have A.P. views of this. I checked those, and I do not think they will show up as much as we are seeing now.

DR. BRENNAN: I would more or less have to go along with what was said. Looking at this plate, it does seem to show there may be some calcium deposit right there at the base of the tibia. I do not believe you can appreciate that at all, looking at the screen.

MODERATOR MILNE: You let me down. I told this practitioner we would have a firm diagnosis. [Laughter]

Next this is the carpus. I think this picture illustrates how a fracture might be missed if you are not on the ball, and if you are not blinded or misguided or misled by other pathology present.

Again, I think in fairness to the panel, I am going to ask them to come up here and comment on this one. It is your turn, Dr. Delahanty. I think you will see it much better on the actual plate than you will on the screen.

DR. DELAHANTY: I think right down here at the head of the metacarpus is a nice little line coming in the front edge, and a little area of decreased density right through here. Those in the back could certainly see this hook, and in the radiograph is another faint shadow. Granted, there are other changes on this film that bear some looking at. There is irregularity here along the front of the radius; it is old; it is smooth, no really hot spots on it. One suggestive area right there, and then another little sharp spurring, that to me is an older lesion. Maybe this is the background. A little alteration in the use of the limb; it avulsed and caused the fracture in here.

MODERATOR MILNE: Dr. Brennan, would you like to come up and have a look and see what you would do about treating it?

DR. BRENNAN: On this radiograph here it exhibits quite extensive calcification. One would have to ask if the animal was showing excessive pain. I would certainly feel that surgical removal of that piece could be done, plus the excessive calcification scraped away. Is there any particular history with this horse, that you know of?

MODERATOR MILNE: I am sorry, I haven't much history.

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It was told to me but, candidly, I am getting so knee deep in x-ray plates, I don't know. Bruce, can you confirm?

DR. BRUCE FARQUHARSON [Phoenix, Ariz.]: This picture is three months after the original film was taken. This horse evidently was cast in the stall. The owner took him out, galloped him. That morning he pulled up lame. The original film was taken in June and this one was taken in August.

DR. BRENNAN: With that length of time elapsing, one would have to think that you would have to go along with nature, then, and keep your fingers crossed. It is quite probable he is going to be an unsound horse. That would be my opinion.

DR. DELAHANTY: With three month's spread between initial injury and this, yes. With this one single film, you might say that it is healing, but I think all of us here will accept the fact that, if you stuck your neck out on one single view, until you took a pair of obliques to get the while size and shape of the particular fragment in here, you would be on this ice. The hook that is below it on the anterior aspect is very smooth, quite old.

That brings up an interesting point. A set of films was sent around to radiologists across the United States—human radiologists. There were fifty films. The same set was sent back to them two years later, in a little different form, the same films, the same history. The correlation between the original interpretation and the one two years later was not better than 50 per cent. [Laughter]

MODERATOR MILNE: You mean this is just a racket! [Laughter] I am going to change over to another one.

[Slide] This is one of my own. I throw this picture in because to me it was most interesting. This horse was sent down to me by a practitioner to see if I could stop the hoofs sloughing off. She was fifteen years old, a broodmare thoroughbred. I examined the foot. The owner confessed that two weeks ago this mare had got a nail. He had taken it to the farrier, and the farrier pulled the nail out and got some pus. I think what happened there, of course, is that the wound closed over. We have pus in there and the bone is just shot.

The thing that interests me is this: that took place in ten days to two weeks. Look at the far side of the plate; the bone is eaten away. Believe me, that is true. I have got the thing pickled. [Laughter]

Dr. Delahanty suggests that perhaps the nail might have caused the fracture; yes, a definite possibility, although the mare was going pretty well, he said, until a few days later it started to blow up. It is an awful sight seeing it slough off; when you press on it, you get a squelching of the coronary band.

Do you remember that last year I asked the opinion of some of you fellows on this plate here? Not many of you were able to

help me. Now, boys, I have you here and I want a good story on this.

As Dr. Reed said yesterday, you don't meet a condition for years and all of a sudden you get two or three of these. This is in a thoroughbred yearling stallion, with amorhphous calcified mass over the femoral attachment to the tibia, bilateral, no pain, no lameness. I have had it in a standard-bred brought in for roaring. The owner said, "What is that bump?" It looks as if it was on the fibula. We called it a chondroma and wondered if it was.

We have angular shots showing it starting off very small. I know Dr. Delhanty had one case, and I know Dr. Bruce Farquharson had one. I know of two cases operated on, one by my colleague, Dr. Blood in Australia. I said, "How did you get along?"

He said the horse was worse off than before. "He was lame when I got through with him." Dr. Delahanty, I know you had one.

DR. DELAHANTY: Mine was lamer than hell, too. I had one of these presented to me. The history was rather interesting. The lesion had apparently migrated across the stifle joint. This is O.K.; maybe it did happen. The owner was adamant, wished it to be taken out.

We went in, removed the calcified, amorphous mass. It even involved the entire head of one of the flexor tendon muscles; I just cannot remember which one it is that attaches right there. It was a little bit lower than this one. We actually removed the head of the muscle, the origin of the muscle, took off the belly and closed it up. Of course, we played around with the lower cul-de-sac of the femoropatellar joint capsule. It did heal up, but the colt did not amount to anything. We sectioned it and there was white, chalk-like material. If you cut through with a knife, chalk came off on the knife. We sent it around to such people as the Armed Forces Institute in Washington, D. C. We could not get a diagnosis anywhere. I wish somebody could help me.

MODERATOR MILNE: Dr. Proctor, let us see what you have to say about it.

DR. PROCTOR: Of course, half knowledge is one of the things that gets us in trouble. I had something very similar. It developed that some gentleman had used one of the sclerosing agents in an attempt to sclerose the head of the fibula in the articulation there with the tibia. From x-ray appearances, it was almost indistinguishable from this mass here. Needless to say, the sclerosing agent did not get the job done.

Not that I am an anatomist, but going into this matter of removal of the head of the lateral digital extensor which arises from the head of the fibula and the femorotibial ligament, one of the things that we did there, in an attempt to alleviate this

condition, to do an old stringhalt operation. We felt that the lateral digital extensor, from its point of origin, might be one of the etiologic factors in this fibular condition, when we first started. I have done several of them. You do get some alleviation of symptoms, but I would not recommend it as a routine procedure.

MODERATOR MILNE: Dr. Riley, please!

DR. RILEY: I have a slide up there of just the identical thing. I was going to ask you fellows about it, so I won't say anything. It definitely is not a myositis condition.

MODERATOR MILNE: Dr. Brennan!

DR. BRENNAN: No comment.

MODERATOR MILNE: I am going to put one more on this slide apparatus, and I am going to ask each of the panel members to show some of their own slides, and help us point out various things.

[Slide] Dr. Delahanty, you remember I was telling you about a horse that was not lame, you could not get any pain reflex. That is the one that made a monkey out of me.

Would you like to comment on this one?

DR. DELAHANTY: One thing that one of my past teachers taught me that I think is rather important in articular lesions around the joint is that, if there is a fairly sharp line of demarcation, oftentimes nature will carry the articular cartilage over this quite a way. He astounded me many times in looking at a film much like this. He would say, "Don't worry about it, he will be all right. Look how smooth that line is." Almost invariarbly he was correct. When do you condemn them and when don't you? You learn, as you stick your neck out, to be a little bit more conservative each time you get it slammed.

MODERATOR MILNE: Want to comment on this, Dr. Brennan?

DR. BRENNAN: I would say if the animal were race horse sound, I would leave well enough alone, just as was shown in the pastern slide earlier, where you have one with rather extensive calcification, and yet they are making out all right. Just look at it, at it, hold your tongue and wish the man well. It is amazing to me the way some animals are able to race with extensive lesions in the knee and in other joints. Some of these horses that are aged and showing just a little stiffness in the joint, I feel you can prolong their usefulness if you use some of the corticosteroids in the joint. But where there are no signs of inflammation, just keep your syringe in your pocket and let them go on their way. That is the only thing I have to say. To do nothing sometimes is the best. In this instance I certainly would say that is the case.

MODERATOR MILNE: A question I am often asked is, can you get rid of it, put anything on this? It does not look too solid? Have you some advice, Dr. Riley?

DR. RILEY: I do not advise any surgery on it unless the horse has been troubled very much with it, and the prognosis for outcome is very bad. You have a lot of damage that does not show on the x-ray. If you dissect out one of the knees that has as much showing as this on the film, you will find a great deal more in there that, even if you took that out, I doubt if you would overcome the inflammation.

MODERATOR MILNE: I am inclined to agree; as long as he is going sound, I am not going to interfere.

DR. PROCTOR: The thought that came to me is I don't think anybody can make an intelligent evaluation, as we said here several times before, by looking at a single x-ray. I wonder if we as equine practitioners could not sort of evolve what would be a logical, standard procedure for x-ray examination of a carpus, for instance.

My personal feeling on the thing is, if you are going to do a decent job and get these secondard manifestations that Dr. Delahanty is so wrapped up in [Laughter] the least we can do is take an A.P., lateral-medial, lateral oblique, medial oblique, and a picture at absolute right angles to the carpus.

My personal feeling is that any type of x-ray examination that does not involve those five plates leaves something to be desired. What is your reaction to that?

DR. DELAHANTY: I agree wholeheartedly, Dr. Proctor, we all are sticking our necks out. We need a standard protocol. I think we are all aware of the fact that each case may present a little different facet. We may take a set of standard views. Many times another different angle brings the thing out from a shadow to a clear line.

I wonder if Dr. Leroy Johnson is in the room. I would like to have his comment on radiotherapy in some of these with periostitis. Some of the films I have seen of his work, before and after, are absolutely amazing. I would swear up and down it is not the same leg. Is he here? [Absent] It is too bad.

MODERATOR MILNE: O.K., would you like to take over, Dr. Delahanty?

DR. DELAHANTY: I am a stickler in a way, as is Dr. Proctor. There are a lot of things to be learned from radiology, in the sense they will teach us a lot as diagnosticians. They can lead us astray very easily. Some of the films I brought together here today are with that very thought in mind. I think, as we all take a first quick look at this particular film, we are all met with a nice big sidebone; I do not have to point it out; and a shorter one on the other. But I don't think that this is the entire story on this horse. Perhaps you can see in the back, starting to hook up here in the corner of the navicular, a cloudy anterior joint space, a little bit of sclerosis. As I put it down in my own book, which is which? Is it sidebone? Is it a case of navicular disease or both together?

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I grant you I put you at a decided disadvantage. I cropped these down, have taken only single projections to put the thing across. If you appreciate my point that we are trying to see some of the less obvious things, that is all I wish to accomplish.

This is pretty easy one to see. Here is the corner tooth, the second premolar. Here is a nice big cavitation. You can tell it osteomyelitis. I think most of us are well aware there is a fracture of the ramus. If you clean up the infection, this is going to heal. The prognosis is a little different, when we accept the fact there is a fracture. So, look the whole thing over and try to get the history together on it.

I had this film circulated amongst a group of sixteen students a couple of weeks ago. We batted it around for two hours. Sure it is ringbone; no question about it, but how did it start? Is it a true ringbone, or does this little shadow in here represent an area of new periosteal bone, and we have a fracture come off there? It went back and forth.

This one is probably a little difficult to see. Perhaps I can orient you a little. This is the ramus of the jaw. This is the esophagus; a barium study in here, and I was pleased as Punch on this one. I made a diagnosis of cervical choke. The colt was only four months of age, just going on hard food. We took him off, fed him fluids; took the muzzle off in seven days. We had to let stuff out of there, he was starting to eat wood shavings, and he was choked again. We missed the diagnosis. Here is a place where x-ray will not make a diagnosis. This colt had a cicatricial stenosis down in here which was aplasia of the mucous membrane of the esophagus. This was shown on postmortem. The only way to look at this is by fluoroscopy and, frankly, I don't want to get around to that stuff.

We are all looking at the hock. This is a big old draft mare, with ankylosis of the hock; sudden lameness. She has a kick wound, with a nice chunk out of the lateral splint bone. Clinically, the diagnosis was easy to make. Here is a little wound, a little bit of drainage, a little bit of blood, not too sore. You look at the film and you find another source of trouble.

These are the kind I really like, trying to work around the vertebral column of the horse. Frankly, two lesions in here. The cap of L2 is knocked off. Perhaps you can see a fine line. If you look in the neural arch, L2 is nice and smooth. We come back there at L3 and see this little point sticking out.

On postmortem there was compression hematoma, with paralysis behind. It is a challenge, but in a way it is fun.

DR. BAKER: What is the size of your machine?

DR. DELAHANTY: This was taken with a Picker 500 MA, 100 kv. 30 milliamperes second. We shot it at 300, tenth of a second, with a grid. This isn't a Picker portable or G.E. portable type

of technic, I must admit. All the rest of the films were taken with a 15 milliampere portable unit.

MEMBER: How old was that horse?

DR. DELAHANTY: Four years old. The history on him was very vague; we did not have one. He was shipped in from a racing stable. The trainer had not come.

This next plate brings out the point Dr. Proctor made so nicely. This tells us nothing. Here is the fetlock, cannon, nice area of grayness, nice smooth layer over it. Here is a beautiful subpericosteal hematoma with organization and calcification.

[Slide] If you look up in the corner, there is a nice gray line going across, with the corner of the navicular bone knocked off. Usually you will find, if you have fairly good films, evidence in more than one projection, of fractures. We all know the difficulty there is in getting films at two different angles on a fracture case to make the diagnosis a little more certain in our mind.

MODERATOR MILNE: Give us any help on the etiology of those fractured naviculars? May we get them in horses that have not previously shown navicular disease? Have you any clue? I asked Dr. Guard about two years ago, and he thought it might be direct concussion similar to what causes a fracture of the os pedis. Have you any comment on that?

DR. DELAHANTY: I failed to say this animal does have navicular disease. As I wrote the case up, it was fractured navicular bone plus navicular disease.

MODERATOR MILNE: Do you agree you can get them without navicular disease?

DR. DELAHANTY: Very definitely.

MODERATOR MILNE: Those are the ones that confuse you.

DR. DELAHANTY: Very definitely.

[Slide] It is always confusing to work around the head. If you take time to follow out each tooth, you can get a diagnosis. I do not know any way to really bring them out without a great deal of distortion. Look them over carefully.

This plate is a pretty good hock lameness to me, and that is all. It shows one hock joint space here pretty well, and another one, with complete loss of the articular surface, perhaps a suggestion of it right in here. To me, this brings out a very important point. Many of us, in our diagnosis of spavin lameness, look for the median lesion here. I think I have found sufficient evidence radiologically to warrant me to look in the articulation and practically disregard that which occurs out here in the periphery.

MODERATOR MILNE: Thank you, Dr. Delahanty.

Dr. Proctor, are you ready?

DR. PROCTOR: It is always my good fortune to follow some chap that is exceedingly glib, and one of the best lecturers in the country, not to mention one of the keenest veterinarians, but

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I got even with him the other day. I kept him up until six-thirty in the morning, treating horses, and we were sober [Laughter]

This is a slide that we saw yesterday, Dr. Reed's picture of the animal with the so-called pseudo-ringbone or upper luxation or subluxation, whatever you want to call it. I think this is very important. We run into it quite often. Firing gets a lot of credit for curing these so-called ringbones, which they aren't at all. As I Isay, this is a very common entity and one that could be distinguished. It definitely is not ringbone, and a good prognosis can be given.

However, I have seen horses racing with as much as a 5-degree variation in the angle of the foot. I find it is not wise to try to make the angles of the two front feet meet too well. This is the way nature has built the foot, and I think it is wise to leave nature alone.

This is the honest-to-goodness ringbone that involves the articular surface. This animal is not sound and will never be sound.

I forget whether I showed this one to you yesterday. It is one of my favorite x-rays. It is of a Gelding, 17 hands high. He has a pair of beautiful sidebones, but the thing that eventually limited this animal was the extreme pigeon toe, off-center distribution of weight. Notice the compaction of the joint here, the opening of the joint here and here. To me, it was always a marvel that this animal could go on a 20-mile chase and come back sound at all. Nature caught up with him in the final analysis. I think the only reason he lasted as well as he did was they did not put him into heavy work until he was five or six years old.

[Slide] By the way, all of my x-rays are with a Picker 15 milliampere. This is the jaw of a yearling colt. The ramus, as you see, is badly broken and overriding.

I got busy with the thing. I did not have to do an open reduction because the colt had done that when he hit the fence. We drilled four holes through interlaced with wire. I was not satisfied with the immobility.

Having seen it done in the dog, I took an ordinary intramedullary pin and went in opposite the little papilla in the bottom of the mouth, just beyond this small papilla with an ordinary intramedullary pin, and eventually was able to draw the pin, with an ordinary ball-peen hammer right down the ramus of the mandible, and we did get good healing.

DR. RILEY: Leave the pin in?

DR. PROCTOR: I could not get the pin out. I would like to have taken it out but I couldn't. It is still in.

[Slide] Again we get to the technic with this 15 milliampere. I do think you can see this fracture. This is about the eighth or ninth thoracic rib coming off the vertebra. The picture was diagnostic, due to the fact the animal showed a great deal

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of humping of the back. It was not an exceedingly valuable animal. We went ahead and destroyed it.

[Slide] This is a fracture of the os pedis. I feel that the only significance here is the fact that it does not involve the articular surface. This animal, with a bar shoe and six to twelve weeks rest, depending upon the progress, will go on and race successfully. In the event he does not, a single digital neurectomy on that one side will make him sound. I have many such.

I wanted to bring out the fact that this is not a serious condition and pretty nearly guarantees the man that the animal will get back to the races.

[Slide] I, too, have had navicular fracture. It could be that I have got rarefaction in the front, and dipping. In answer to the point Dr. Milne brought up, I feel in some cases these are definitely traumatic, because until recently I never had one in anything except a walking horse or saddle horse. I think the long toe that they have in those breeds predisposes to navicular fracture.

[Slide] Unfortunately, this does not show up quite as much as I would like. This is the x-ray picture of an animal with an acute suspensory condition and sesamoiditis. If you will notice the difference in the A.P. view of these two sesamoids, the suspensory is distended out this far, and there is some increase in the joint fluid over on the left. This animal was acutely lame. We suspected sesamoid fracture; it was not. But I feel that the recovery period on these is almost as long as if it were a fracture. This animal has to be let up on for four to six weeks, in my experience. He should be re-x-rayed in that length of time because possibly this suspensory will pull loose from it and you will get a fulminating condition.

[Slide] This is proximal sesamoid fracture. I would like to summarize what I think we have brought out during this meeting, that these things do respond to surgery, and in almost 70 to 75 per cent of the cases.

The distal fracture is a much more difficult surgical approach, and the percentage probably drops well below 50 per cent of the animals that go back into training. Dr. Jenny and Dr. Raker have done a splendid job and Dr. Wheat, also, in figuring out the surgical approach to these things.

[Slide] Another distal sesamoid fracture. This one would not be amenable to surgery. This is an old one, and there is beginning calcification. I would not recommend this animal for surgery under any conditions. Further I feel that this would bring out the point I tried to emphasize a moment ago, that nature, in attempting to get this thing back into orbit, so to speak, is going to put down enough periarticular calcium, that this animal's racing ability will be very much in question.

[Slide] Here, too, is an example of why, as we know, x-rays

are a series of shadows, and we discussed the advisability of plates from all angles. I also would bring to your attention that these conditions are never static; they progress.

This animal is a foal; as you see, there is quite a lot of cellulitis on the front of the ankle. It was a disfiguring thing. The gentleman was much upset by the possibility of not being able to sell this nice filly.

[Slide] Six weeks later, when this picture was made, there was an area here of calcification, almost as big as a small pecan. What I am trying to bring out is a negative picture in an acute condition. Sometimes you can take your pictures too soon. In other words, there is a calcium lag, but in six or eight weeks it was obvious what the condition was. It took that long for the process of fibrosis and scarification or scar tissue fibrosis to get around to the place where it was obvious on x-ray.

[Slide] This is an animal that had lipping of the distal end of the radius and the proximal end of the ulnar carpal, and this animal was quite lame. He received 1000 r of gamma radiation, and it was repeated in six weeks. This is the postradiation picture.

[Slide) This was not bad. It cleaned up very nicely. I feel there is a definite use for gamma radiation in these beginning calcifications. The way I try to explain it to my clients is, if you had a jagged icicle and held it in your hand for five minutes, you would have an egg-shaped piece of ice. I believe we can, most important of all, stop the process of calcification. To a certain limited extent you can stop resorption.

[Slide] This plate is of a much later type of calcium deposition than that which Dr. Delahanty showed previously. We have subperiosteal hematoma, sequestration, absorption and calcification. There is a tremendous mass. The soft tissue picture would extend out a full half inch beyond this area that is calcified. Because it was so close to the joint surface, the prognosis was very guarded.

[Slide] This one I thought was worthwhile. There was actually about an eighth of an inch separation there, and the head of the fibula is adducted to the tibia. This x-ray was made because the animal showed the symptoms that Dr. Lundvall described in fibular lameness. I, for one, feel that when the symptoms and the x-rays correlate, the entity must be recognized.

I had one other slide that showed a fibula with a double fracture and a complete movement to the side. As I say, sometimes I do not pay too much attention to the x-ray or symptoms, but when they correlate I think you have to recognize them.

MODERATOR MILNE: Thank you, Dr. Proctor.

Dr. Riley, are you ready?

DR. RILEY: The thought struck me, looking at the fracture of the distal sesamoid, some veterinarians may have had the exper-

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ience I have had in blocking them off and getting a complete disappearance of symptoms. It is a great temptation to keep the horse trotting up and down, to demonstrate your point. I would advise you to stop him right away, because the next day he might be exceedingly lame, much more so than he was before.

If another x-ray is taken, you may find fracture of that bone. It is quite brittle. When you suddenly turn him loose, trot him up and down, it leaves you open for legal action.

[Slide] This is a bone cyst. I don't know if this is very clear. This is a 10 milliampere machine. I don't know what the significance of that is in a bone. I have asked our radiologist and the answer he gave me is that they have them in humans and they don't actually know. I thought I would bring it to this meeting. Maybe somebody else has seen that same sort of thing. Is there anybody on the panel or in the audience who has experienced this?

MODERATOR MILNE: I have seen them but don't know what they come from.

DR. RILEY: Ever see a horse lame with them?

MODERATOR MILNE: No.

DR. RILEY: In this horse there may have been some relationship between the inflammation in the tibia and this inflammation. If you took a picture of a joint and this showed up, would you call it sound or unsound? I would call it sound, if it wasn't lame.

[Slide] We have been looking at that mass of calcium in the fibula. One of the veterinarians, whom I am sure is here, Dr. Lickfeldt from Plymouth, sent me this to evaluate. This, of course, is myositis ossificans, with the plating you get around the fibrous structure of the muscle. When you operate on one of these you find this is in the form of a circular plate. It is a little different from the inflammatory reactons you get around the fibula. I also happen to have a fibula picture, if we get that far. There was no lameness in this case, as I recall it. At the time my opinion was asked, I advised no surgery. I have since then run into them in sesamoiditis and tendinitis where there is definite impediment of gait, whereby the horse would advance the leg to normal plane of flight and would be stopped about halfway through; the foot would be jerked once or twice. We simply went in and resected, according to the article Dr. Adams wrote.

If it were not causing any lameness, I do not believe I would touch it, maybe advise another x-ray and re-evaluation in six months' time.

[Slide] This is a Shetland pony with fractured large metacarpal, one of the common fractures they get. The leg was broken on Saturday in a pasture in Indiana. We received the pony on Tuesday. The fracture was compound. A triangular, sizable segment of the large metacarpal was lost at the time of the fracture, and they did not bring it with them. Had they brought it with

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them and kept it clean, we would have ground it up and put it in there.

The reason I present this film is to point out something that we now do. We set this, of course, with the transfixation we have done for years, and gave it a little extra time, sixteen or eighteen weeks. We took the fixation off. I had it in another cast, a temporary cast and, of course, we got nowhere. I talked it over with Dr. Brinker. We decided to try a canine technic which we will show you.

[Slide] We put in a Jonas' pin. This was a three-year-old, pure-bred pony that the man had paid fifty-some-odd hundred dollars for, and he said he didn't want to give it up at any cost. We still had to take care of this area here.

We had resected a rib under aseptic conditions from a horse. I might go back a little on that. We resected the rib, aseptically skinning out the periosteum, removing the sections of the rib and putting them in glass jars and deep freezing them; sending pieces of the rib to the laboratory for bacteriological examination. A day or two later we got a report from the laboratory "hemolytic Streptococcus." We said, "We broke our sterile technic." Unfortunately, we put that horse on out. We waited a few days and another horse came into the clinic that I thought should be destroyed. We took double precautions. We were certain that our technic was O.K. We had no break in our asepsis. We took quite a few ribs off that horse for a bank. They are now in the deep freeze.

We set the leg, put the pin in, the same as you would for small animals. We ground up pieces of the rib and compacted them in the skin and around the fracture site and sewed it up; put our plaster on. This is one of Dr. Thorpe's walking casts. That pony did very nicely.

[Slide] There is the fracture. We left the pin in, of course. There is the well we got from the fractured chips. A couple of weeks after we finished, we decided to run some of the chips through the laboratory. They came back "hemolytic Streptococcus in the marrow." We kept this particular case on penicillin for at least ten days to two weeks. I feel, based on this and a couple of other cases, if your technic is fairly accurate, you do not get any contamination, but how do we know the horse does not have a resident type of infection, or at least harbor some of the bacteria in the marrow? It is an interesting question. With some of the research we are thinking about doing, we hope to answer that.

QUESTION: What is the double line?

DR. RILEY: That is the old pinhole. When we pull the pins, all we do is put in a little penicillin, bandage, and in a day or so they are healed over.

[Slide] There is a lateral view. There is a little cavitation here but it never bothered that pony. Of course, we had to put a corrective shoe on. He spent so many months in a cast that he

just was getting nowhere, as far as support. The muscle came back, however.

Since then I have repeated this process in a ten-day-old filly. I did not need to use bone chips. She wasn't a compound fracture, just a transverse fracture. They are the hardest to set. The diagonal one sets much better. We are going to take x-rays of that filly every five or six months and see if the pin gravitates downward in the joint. I took some about ten or twelve weeks after I set the second pin in, and it hadn't moved. She is running around at the college farm. We are going to dedicate her to science for at least a couple of years and not sell her.

DR. PROCTOR: When you put an intramedullary pin in a ten-day-old foal—

DR. RILEY: Not intramedullary but a Jonas' pin. It is a cylinder with a pin in it, and it has a spring. The outer sleeve, stainless steel, has a spring. You push the pin up in there, compress the spring and lock it in. You take a reamer and go up here, just the size of this outer sleeve, the sleeve comes to here, make a seat for the dorsal portion of that Jonas' pin. Holding your fracture at right angles, in open reduction, you line up another hole down through here with a smaller reamer that makes it large enough. The trick is to get accurate measurement of that bone and your pin, so you have opening from here to here; the pin will fit in exactly. You do not want it to gravitate. That is the experience that small animal people had. They passed it on, so we did take special care. As soon as you release the spring with a key, that pushes it right down in there.

DR. PROCTOR: The thing you must keep in mind is, if the pin or reamer happens to destroy the epiphyseal line, you are not going to get any more growth.

DR. RILEY: That is right. So far there has been no swelling, no change in the growth rate between this leg and the other leg. The second one was put in seven months ago.

DR. PROCTOR: You got normal growth?

DR. RILEY: So far, yes. Of course, the other pony was three and one-half years old. I have had several pictures sent back from Indiana where they are using him as a stud.

DR. PROCTOR: You don't have any problem in the older horses?

DR. RILEY: That is the point. This is actually an experiment. We will see how it does.

[Slide] This one had a little chip out here. This horse was a standardbred. The history was that it was showing lameness periodically. It had received four or five injections of, I don't know whether McKay's or not, in the fracture site. Judging from this one film, I would say that is not the treatment of choice. We were asked about removal.

At that time I had heard about several cases where they

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had attempted to remove the fractured segment of the fibular bone, similar to removing the fractured splint bone, and had difficulty in muscle closure, fascia closure, and had herniation of muscle. I did not do anything with this. I just present it as a matter of interest.

You will notice the difference in consistency, on x-ray, of the calcium deposits here as contrasted with No. 2 slide where you had myositis ossificans. This is definitely inflammatory, and I do not know what causes the calcium shields in the other. I believe trauma starts it.

QUESTION: Any history as to how long since the horse had been injected with a counterirritant?

DR. RILEY: I took the radiograph in August—this was probably two or three summers ago—and he had been injected out East not more than eight or nine weeks prior to that. That is circumstantial. He may have had it before he was injected.

DR. BAKER: The reason I asked, I wondered if there was spread of iodine compound.

DR. RILEY: You could palpate. This particular piece was definitely under the skin. I have had one or two since then that were egg-shaped and hard.

[Slide] Here is a quarter-horse with a kick on the cannon. We used just ordinary antiseptic, sent it home, hot packs and so on. It healed up fine but continued to become enlarged. So I had it brought back for x-ray. This was November. This happened in October. Here we find cavitation with a small sequestrum. If you look at the date on that, November 24, it was right after deer hunting and just before Thanksgiving. I am watching and waiting on that case a little bit. If necessary, we will tap this; go in and bore it and ream this out with a curette. Maybe it will heal. Ordinarily we would open it up, clean it and use varizyme.

DR. BURGER: I opened one and chiseled.

DR. RILEY: Does it tend to recur?

DR. BURGER: It hasn't over three or four years.

DR. RILEY: I remember one time Dr. Gadd from Maryland said he had done some of them. I have been encouraged to try them.

[Slide] This was a horse brought in to have a roaring operation. I throw this in as more or less of interest. We palpated the larynx, as I usually do; for what reason, I do not know. I never could tell whether there was atrophy of the cricoid muscle. I noticed how hard it was. I took an x-ray, and the whole thing was calcified.

I think that is about all I have.

MODERATOR MILNE: Thank you, Dr. Riley.

We are running a little bit behind. We will turn the program to the Chairman. I would just like to say thanks to the panel. [Applause]

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CHAIRMAN WOODCOCK: Thank you very much. The truth of the matter is, when Dr. Milne took over the operation of this panel he came to me and said, "Don't worry about the time because we won't be able to use up more than an hour, anyway." So, he wound up being late, too. But it really is a fine tribute, gentlemen, to a group of men taking over and handling such a very, very interesting panel. As I told you before, it was a trial balloon. If this is any indication, it will be a permanent fixture because I am sure everybody was very interested and enjoyed it extremely.

REPORT FROM GRAYSON FOUNDATION

MR. CLARKSON BEARD

Mr. Chairman, Members and Guests: It is a pleasure to be present with you again this year and to review the work and plans for the Grayson Foundation.

I first want to compliment all of the officers and those that had the work of organizing this meeting. I thought last year was a fine meeting, but this one is even better. I also want to compliment the members on their attendance at these meetings. I would like to suggest a research program which would be a little bit out of my line. I think we ought to institute research into a method of therapy for the posteriors of equine practitioners when they attend the annual convention. [Laughter]

Before we get into that, I think I have one method of therapy, and that is to be just as brief as possible. I want to give you just a quick outline of what we have been doing this year, some of our thoughts, and our need for some money to get some work done.

The work reminds me of a jigsaw puzzle. We are getting some of the pieces into place, and it is making a sensible and logical and workable plan. Other areas may be coming into place; and there are some areas that are still just stray pieces around the table. Our bulletin contains practically all of this information in more complete form. It has been sent out to the Grayson members and will be sent out to all members of the Equine Association just as soon as we can get the mailing list typed up and have the bulletin sent out, which should be shortly after the first of the year.

To give you a brief resume of the actual work done, Dr. Roger Doll has been busy with the field testing of the live virus vaccine. In 1958 over 2,000 horses were given this nasal spray. The results are promising but not conclusive as yet. This year 5,293 horses received the initial treatment in the summer; 2,699 of these were mares; 3,487 horses in Kentucky and 1,752 horses in other states. They were about evenly divided as to thoroughbred and standardbred. One interesting phase of this will be with some 500 standard breds treated last week at the Orlando, Florida, training center. All the horses in that training center have been given this nasal spray to get an evaluation of this treatment, as far as influenza is concerned, when they get to the track next year. I cannot speak authoritatively on this, but I know Roger Doll does not expect that to be any kind of a cure, but we do hope that it will alleviate the condition of influenza. It will be a field test to see just exactly where we stand for that phase of it.

At the University of Kentucky a great deal has been done to give us a more rounded program based on the diseases of the

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horse, particularly the viruses. Isolation units are being built under a grant by the Keeneland Foundation, and a tissue culture system is being established. Roger told me not long ago he has one of the viruses growing in the culture system. Grayson extended its grant to include parasite work under Dr. J. D. Drudge, at small additional cost, utilizing the experimental animals fully. This is to help him out, really, on a testing of certain drugs and some screening work. It is not research at this time. We hope sometime to have research work done along this line. However, at present, the screening and testing are keeping him busy enough.

I would like to mention some salient points on the questionnaire sent to the members of your Association last year, last spring. The returns on these were most gratifying and helpful to us. In using these reports, I want to stress that we have to exercise a great deal of judgment. We cannot just go right down the line, A, B, C, D. It isn't that kind of a survey and not intended to be. There will be cases that will change, conditions will change, and a great many factors have to be more or less correlated. But the returns have been of great help to us in more or less planning an outline of work. It has been of great help from the reaction of the various veterinary schools and, also, actually right here on the program at this meeting. As a result we have staked out four major areas of research: in the field of unsoundness of joint, bone and tendon — I will mention a little bit more about that later; in the field of nutrition, which was given very high priority. There is no way at this time we can get into such a vast field; we do not have the money, and I seriously question whether we could really get into it on that sort of basis and get the kind of results we want. We are going to try to approach the nutrition problem in other areas of research. Nutrition might be a factor in the case of certain unsoundnesses.

Then come breeding problems. These included principally the oestrus cycle of the mare; evaluation of stallion semen and, to a lesser extent, and quite definitely lesser extent, genital infection. The latter did not seem to be quite as important to the practitioners as the others. In this field not only do we not have enough money but we also have to get some sort of a plan that will be workable. We have had a couple of projects: one on oestrus cycle of mares, and another one on stallion semen. Technically and scientifically, the results have been fine, but we are not making the headway that we had hoped, or should hope, to do. We must study how to approach this problem. It is amazing to me how little information we have to go on, even in other breeds, in that respect. Work is going on along that line. A group of us in Kentucky is trying to plan something that will be sound and practical. But here, again, it is going to be a question of the funds to get this done.

Now, in disease, principally the viruses of influenza and

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abortion and parasite diseases, we have a pretty well-rounded, operating program at the University of Kentucky.

Other items of interest include general anesthesia, which was given a high priority, and on which we have contacted several of the drug companies. I have been disappointed in their reaction. They simply come back with, "Well, you do the work." I think by plugging around here and there, we will get something done on that subject. Also, right here on this program there were some very interesting items regarding anesthesia. The condition of tied up was discussed this morning. I do not think I need to make any further comment on it. Then leptospirosis and periodic ophthalm a were taken up. Normal blood chemistry and metabolism were not among the important subjects I mention them simply as it seems to me they may possibly be more important than the survey indicated. Every time you run into a question of any research work, you come right down to those normals.

Earlier we had contacted the veterinary schools in the United States and Canada, and we found there was a considerable increase in the number of horses admitted to the large animal clinics during the past five years. In the case of ten schools, they gave us comparative figures. These schools are hampered by the lack of funds, lack of holding facilities for horses, and personnel interested in the work. That is a very general statement and should not be considered to be specific anywhere. The important point here is that, by sheer numbers, interest in the horse is being reawakened in these schools. Some of them are now becoming quite interested. The veterinary schools, with few exceptions, are best equipped to do equine research work. This is particularly true from the Grayson point of view, in that they have the equipment and the staff. If we start getting into the hardware of research, and we have gotten into it, you get a lot of equipment spread around, and then you do not take full advantage of it. We are trying in every way to avoid that. As a matter of fact, our grants now are made in such a way that they do not include it. If a necessary piece of equipment is essential to the research work, it is bought by the institution and in Grayson's name, so that at a future date it can be moved, if necessary. That is a very important point, particularly with our limited funds.

Using this questionnaire and the information we have received from these schools, we asked for limited proposal for studies in unsoundness, where the schools had the facilities and the personnel to do the work. This is with some available funds which we cannot say, definitely, will be recurring. Our limitation was set at $10,000, an arbitrary figure to work with, but one that would give the schools some idea of how we were thinking, with no assurance of our being able to continue without continued funds being available. We asked for studies in any phase of unsoundness, of joint, bone or tendon, or nutrition as it affected those condi-

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tions. So you can see it is a pretty stiff, tight proposal, as far as finances are concerned. I was expecting to get maybe two or three proposals; actually we got seven; two more are doing it. So, we have nine proposals along that basis.

The idea here is to give these schools the funds to go ahead with some of the pathology of these unsoundnesses, with the idea that we can develop areas that we can continue later. It is in the field that seems to be most critical to all the practitioners. That is the approach we are taking on it. I wish it could be more, and I hope we can develop more on it. These plans will be submitted to our advisory committee shortly, probably right after the first of the year, and I hope some action will be taken on them before too long.

By this activity and, also, I might point out, the activity of your Association, a great deal of interest has been stirred up in this field of research, but this has not been backed up with money. Articles have been published in the trade journals, the veterinary journals, and our bulletin is in the process of being mailed out, as I mentioned a minute ago. Our public relations are not adequate, due, again, to finances. We would rather put the money into research work that we can do, but we should not neglect our public relations activities. Contact and meetings with breeder associations have been our best way to reach as many people as possible. Grayson originally started out mainly with thoroughbred horses, although there is no intention to limit activities to this field. We made some pretty good contacts at the start with the standard-bred breeders, and with the quarter-horses. General Kester has been helpful in working along those lines. I think we have made an initial contact there. These groups are interested in getting some research work done. Support of the racing association is a tough one, as they are concerned with more immediate management problems, and they have people constantly on their backs for funds. How I am going to crack that nut, I am not quite sure. We have had donations from two racing associations that are new on our list, and we hope to expand and continue this effort, but I do not think that it is our main source of income right now.

Fund raising is where we are falling down, and this is part of the jigsaw puzzle that we have got scattered around the table, and we have to get together. We have the interest but we do not have the money to follow it up, and without that we are not going to be able to do it. Our budget is now, roughly, $50,000 a year. This should be $100,000, as a minimum, and we could spend that right quick, and spend it well. I feel that actually it could be closer to $250,000 a year and we would still be on good, solid ground. After being up here and listening to these talks here and the need for research, I think that $250,000 might be low. I think this is an important point for you to consider. This questionnaire has

helped us to give priority to these various subjects, as you have indicated to us, and to go at the most important ones first, and then expand our program. That is more or less our thinking on it. If you disagree, I hope you will give me ideas to change it, as we would like to move much faster. We want to get as many individuals supporting this program as we can, at reasonable annual dues. The more people we have interested, that much easier it will be to obtain some of the larger grants. This is one point that is going to be important to the racing associations. If we have more members actively working in the membership of Grayson, contributing, they will be more inclined to do their part. The associations have had the feeling that they have been carrying it themselves, which is true for about three of them, but that is all. Our dues are $50 for annual membership, and $250 for breeder membership, or one with larger interest in horses. Those are business expenses. They are not contributions, and they would not include any larger grants or gifts that individuals, so inclined, might give to us. One thousand members at $50 would give us $50,000. I see no reason why we should not have 1,000 members. We have approximately 150 individual members now. That should be up closer to 1,000, or even 2,000.

My work this year has been to survey the situation and outline the framework of operations which I have more or less gone over here now. I do not lose an opportunity to plug for some money as well, but there is going to be no single person who is going to do this.

There is a lot of talk but there isn't the money.

To make this go, it is essential that groups such as yours, breeders and racing associations, have committees or individuals who will help in getting the necessary information, particularly regarding the importance of financial support and file of their associations. The situation is now that the plans and ideas are coming along pretty well, and we are still open for any suggestions, but there is also this tendency to let the other person support the work, and that is not going to get the job done. On your part, last year I said that the main thing the practitioners could give us was moral support. I still think that is true. You can be our best ambassadors. If we have a program that fills the bill and the need, and you are the people who are going to have to use it, you can not only by your financial support—of course, we would like your $50, to—we would also like to have you urge clients of yours who are so inclined toward the research end of this work, to support it.

When we sent out our questionnaire and emphasized the financial needs we had, we sent out two membership cards telling those who would like to join themselves that we would be only too glad to have them as members, and asked that you take those cards and get either one or two other people to join. We got, I

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think, four memberships back from that. If this membership of 300 could get two that are not members now, it would put $30,000 in the pot. That would be quite a foundation to work on. If you showed that interest, I am sure that others will show the same interest, and we can also interest associations such as the racing people on that basis.

Grayson's plan of operation is flexible to meet the needs of the industry. I want to mention that particularly, as I think some people feel that Grayson has been a little conservative, which it has been, and that there hasn't been much work done, and I think that is true. I also want to point out that the people who formed Grayson did it at a time when they used a great deal of foresight. There was a period there when there wasn't anybody working on it but them, and it more or less held that gap together. Now we are in changing conditions where we can go on and move ahead, and it is up to everybody to jump in behind. You cannot jump in behind something you do not believe in. We can make this into the type of outfit that is going to get something done. It is a question of being practical about this thing.

You had quite a discussion on the 48-hour rule earlier. It was shown that that is not practical, and it is your desire to make it practical. That is, basically, what you are after. The same thing is true in this case. We want to make this a practical plan that will be worked and can be financed and can go on ahead.

The counsel and advice of your organization is essential to fully develop this plan. It is up to everyone with a stake in the horse business to support the work and make these remarks an actuality. We have delayed long enough in this critical field. Now is the time for some concerted action. There is a lot of work to be done, and we should be getting on with it.

Thank you very much. [Applause]

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